The copper tube is ready to connect. The flaring tool is shown above. The other end of the copper tube will be flared in the trench.

Since it's pretty messy down there, they open the valve slightly on the new water main tap and let the water run for a bit to clear any debris.

Putting the sealer on the threads while the water runs.

Oops! The copper pipe is too long…

The copper pipe is cut using a rotary cutter.

Looks like it'll work

The fitting is slid onto the copper pipe and the end of the copper pipe is flared.

Connecting the pipe ends.

Done. Now to check for leaks.

Oops! He forgot to tighten the fitting on the new main!

Leaking stops…

Done!

View of final connection from the sidewalk level.

Once the connection is made and there are no leaks, the trench is filled with gravel.

And every 10 miles of new main, it is customary to bury one construction worker, so that they can be the guardians of the new main for the next 100 years. Poor Jose...I kinda liked the guy.

The previous pics were a connection to the near side home. This copper connection is longer, going across the trench since the home is on the other side of the street.

This is a picture from the new NASA Mars Rover showing life on Mars. It appears the Martians are made of lead and shaped like tubes. If you zoom in [...] you can see the creature has millions of teeth with which to destroy [...] the planet. It also appears that the sur[...] a nice p[...] and looks rather like an Earth kitchen.

Comparison of WDNR Definition to EPA Definition for Select Consecutive System Scenarios

Consecutive System Scenario[1]	Regulated by EPA		Regulated by WDNR	
	With Treatment[2]	Without Treatment[2]	With Treatment[2]	Without Treatment[2]
Mobile Home Park (MHP) (Single Service Connection from Regulated PWS)				
MHP with master meter that does not submeter[3] or charge separately for water.				
MHP with master meter that submeters.[3]				
MHP with no master meter and all residents are billed by regulated PWS.				
Strip Mall within Regulated PWS (i.e., Single Service Connection to Private Property from Regulated PWS)				
SM with master meter that does not submeter or charge separately for water.				
SM with master meter that submeters.[3]				
SM with no master meter and all entities are billed by regulated PWS.				
Hospitals and Hospital Complex[4]				
Hospital *Complex* (multiple structures) with master meter from regulated PWS that does not submeter.[3]				
Hospital *Complex* (multiple structures) with master meter from regulated PWS that submeters.[3]				
Hospital *building* with single service connection from regulated PWS with a master meter.				
Hospital *building* with a single service connection from regulated PWS without master meter.				
Apartment Building Complex or Apartment Building				
Apartment Building Complex (multiple structures) with master meter from regulated PWS that does not submeter.[3]				
Apartment Building Complex (multiple structures) with master meter from regulated PWS that submeters.[3]				
Apartment building with single service connection from regulated PWS.				

1 – Assumes the entity meets the PWS definition in SDWA, and it must be determined if they are a regulated PWS, subject to NPDWRs.

2 –The Federal consecutive system definition mentions 'finished water' and the definition of finished water means it is ready for consumption with or without booster chlorination and addition of corrosion control chemicals (not counted as 'treatment'). If a consecutive system provides treatment to remove a regulated contaminant, I think it is clear that they are regulated. What is unclear to me is whether a MHP is providing 'treatment' if it has centralized treatment for a secondary contaminant (Fe/Mn removal). In other words, should EPA limit the 'treatment' definition to removal of *regulated* contaminants?

3 – See EPA's revised policy on submetering (2003 FR notices embedded).

4 – Chlorine dioxide (ClO2) use in NCWSs: § 141.130(a)(1) states that NTNCWS adding a chemical disinfectant to their water in any part of the drinking water treatment process have to comply with monitoring requirements. Hospitals are using or considering ClO_2 to control *Legionella*. HQ will be developing an FAQ to clarify when a PWS would have to comply with requirements for ClO_2.

REFERENCES

§141.2 Definitions.

Black text: Existing definitions.
Red text: Potential proposed definitions under the LCR revisions.

Combined service population is the number of people served finished water by a wholesale system and is the sum of the retail population served by the wholesale system and the retail populations of all consecutive systems that receive water from the wholesale system, directly or indirectly through the distribution system of one or more consecutive systems.

Community water system means a public water system which serves at least 15 service connections used by year-round residents or regularly serves at least 25 year-round residents.

Consecutive system is a public water system that receives some or all of its finished water from one or more wholesale systems. Delivery may be through a direct connection or through the distribution system of one or more consecutive systems.

Finished water is water that is introduced into the distribution system of a public water system and is intended for distribution and consumption without further treatment, except as treatment necessary to maintain water quality in the distribution system (e.g., booster disinfection, addition of corrosion control chemicals).

Modified monitoring is monitoring that is modified following the provisions of §§ 141.29 or 141.871 of this part. It does not include reduced monitoring, increased monitoring, or monitoring for wholesale systems and consecutive systems in subpart Z except as provided for under § 141.871.

Non-community water system means a public water system that is not a community water system. A non-community water system is either a "transient non-community water system (TWS)" or a "non-transient non-community water system (NTNCWS)."

Non-transient non-community water system or *NTNCWS* means a public water system that is not a community water system and that regularly serves at least 25 of the same persons over 6 months per year.

Person means an individual; corporation; company; association; partnership; municipality; or State, Federal, or tribal agency.

Retail population is the number of people served by an individual public water system delivering finished water directly to that population. It includes both people served at a billing address and people served at locations that are not billed.

Service connection, as used in the definition of *public water system,* does not include a connection to a system that delivers water by a constructed conveyance other than a pipe if:
(1) The water is used exclusively for purposes other than residential uses (consisting of drinking, bathing, and cooking, or other similar uses);
(2) The State determines that alternative water to achieve the equivalent level of public health protection provided by the applicable national primary drinking water regulation is provided for residential or similar uses for drinking and cooking; or
(3) The State determines that the water provided for residential or similar uses for drinking, cooking, and bathing is centrally treated or treated at the point of entry by the provider, a pass-through entity, or the user to achieve the equivalent level of protection provided by the applicable national primary drinking water regulations.

Transient non-community water system or *TWS* means a non-community water system that does not regularly serve at least 25 of the same persons over six months per year.

Supplier of water means any person who owns or operates a public water system.

System with a single service connection means a system which supplies drinking water to consumers via a single service line.

Wholesale system is a public water system that treats source water as necessary to produce finished water and then delivers some or all of that finished water to another public water system. Delivery may be through a direct connection or through the distribution system of one or more consecutive systems.

§ 141.3 Coverage.
This part shall apply to each public water system, unless the public water system meets all of the following conditions:
(a) Consists only of distribution and storage facilities (and does not have any collection and treatment facilities);
(b) Obtains all of its water from, but is not owned or operated by, a public water system to which such regulations apply:
(c) Does not sell water to any person; and
(d) Is not a carrier which conveys passengers in interstate commerce.

Embedded Files: FR Notices on EPA Policy on Applicability of SDWA to Submetered Properties (Proposed and Final); U.S. District Court of Appeals Decision on EPA's Revised Policy; and ASDWA Issues with PWS Definition.
[EMBED AcroExch.Document.7]   [EMBED AcroExch.Document.7][EMBED AcroExch.Document.7][EMBED AcroExch.Document.7]

Lead and Copper Rule Revisions

* Lead and Copper Rule promulgated in 1991
* Revised in 2000 and 2007

Long-term Issues	
Partial lead service line replacement (LSLR)	• Engaged SAB (2011), and NDWAC (2011-12) • Evaluating revisions to the LSLR requirements
Sample Site Selection	• Evaluating revisions to the criteria to better address the latest information about lead sources
Tap sampling	• Evaluating different protocols for collecting tap samples for lead and copper
Measures to ensure optimal corrosion control (OCCT)	• Evaluating OCCT requirements to better ensure optimal corrosion control and effective water quality parameters monitoring
Copper	•Evaluating approaches to better address copper
Reduction of Lead in Drinking Water Act	•In corporate changes new definition of "Lead Free" from Lead Reduction in Drinking Water Act

17

Lead and Copper Rule Revisions Outreach and Consultations

* Stakeholder meetings October 2008 and November 2010
* Environmental Justice Stakeholder meeting March 2011
* Science Advisory Board 2011 review of partial lead service line replacement (PLSLR)
    - PLSLRs have not been shown to reliably reduce drinking water lead levels in the short term, ranging from days to months, and potentially even longer
    - Additionally, PLSLR frequently associated with short-term elevated drinking water lead levels for some period of time after replacement, suggesting the potential for harm, rather than benefit during that time period
* NDWAC consulted in 2011
* EPA intends to publish proposed LCR long-term revisions in 2013

18

1

Reduction of Lead in Drinking Water Act

- Amends SDWA Section 1417 – Prohibition on Use and Introduction into Commerce of Lead Pipes, Solder and Flux
  - Modifies the applicability of the prohibitions by creating exemptions
  - Changes the definition of "lead-free" by reducing lead content from 8% to a weighted average of not more than 0.25% in the wetted surface material (primarily affects brass/bronze)
  - Eliminated provision that required certain products to comply with "voluntary" standards for lead leaching
  - Establishes statutory requirement for calculating lead content
  - Effective 36 months from signature – January 4, 2014

19

Reduction of Lead in Drinking Water Act
New Lead Free Exemptions

- Exemptions to the prohibition on use and introduction into commerce provisions in 1417(a)(1) and (3)
  - 1417(a)(4)(A)
    - One exemption is for "pipes, pipe fittings, plumbing fittings, or fixtures, including backflow preventers, that are **used exclusively for nonpotable services**, such as manufacturing, industrial processing, irrigation, outdoor watering, **or any other uses where the water is not anticipated to be used for human consumption;**"

  - 1417(a)(4)(B)
    - Another exemption is for "toilets, bidets, urinals, fill valves, flushometer valves, tub fillers, shower valves, service saddles, or water distribution main gate valves that are 2 inches in diameter or larger

20

2

Reduction of Lead in Drinking Water Act
Key Revisions – Definition of Lead Free

- 1417(d) Definition of Lead Free
  - Revises the lead content requirement from not more than 8% to not more than a weighted average of 0.25% lead when used with respect to the wetted surfaces of pipes, pipe fittings, plumbing fittings, and fixtures [1417(d)(1)(B)]
  - Provides calculation procedure for determining the weighted average lead concentration of a product from the components that make up the product [1417(d)(2)]
  - Eliminates 1417(d)(3) – which requires certain products (plumbing fittings and fixtures) to comply with standards for lead leaching (NSF/ANSI Standard 61 Section 9)

21

Reduction of Lead in Drinking Water Act
Key Revisions – Effective Date

- Effective Date of January 4, 2014
  - Amendments become effective at the same for the use prohibition in 1417(a)(1) and the introduction into commerce prohibition in 1417(a)(3)
  - A product introduced into commerce legally on January 3, 2014, can't be used in the installation or repair of a PWS or residential or non-residential facility providing water for human consumption on January 4, 2014
  - Potential purchasers that could be affected by the lack of a staggered effective date include: plumbers, plumbing product retailers, developers, schools, and water systems
  - Back inventory that does not meet 0.25% lead free calculation cannot be installed after January 3, 2014 unless it is exempt from the prohibitions

22

3

Bibliography of Selected Manganese Publications Related to Drinking Water Exposures and Infants and Children & More

Haynes E., P. Ryan et al. (2013). Assessment of personal exposure to manganese in children living near a ferromanganese refinery Sci Total Environ. 2012 June 15; 0: 19–25. doi:10.1016/j.scitotenv.2012.03.037.

Airborne exposure to manganese (Mn) can result in neurologic effects. Stationary air sampling is the traditional technique to assess Mn exposure for communities, yet may not accurately reflect children's personal exposure. The goal of the study was to characterize personal exposure to Mn and PM2.5 in a cohort of children ages 7–9 years residing near a ferromanganese refinery.

A subset of children living in non-smoking households ages 7–9 enrolled in the Marietta Community Actively Researching Exposure Study during March–June 2009 and 2010 were invited to participate. Blood and hair were collected and analyzed for Mn. Participants wore a PM2.5 sampler (Personal Modular Impactor) for 48 h. TWD was based on time spent at home and school and the distance of each from the refinery. Stationary outdoor air sampling was conducted 8 km from the refinery using a Harvard-type PM2.5 impactor. The relationship between personal Mn exposure and TWD was examined by multiple regression adjusting for stationary air Mn concentration, wind speed and direction, and precipitation.

Complete personal air sampling data were collected on 38 children. TWD ranged from 4.7 km to 28.5 km with a mean distance of 11.1 (4.7 sd) km. Mn concentration in personal air samples ranged from 1.5 ng/m3 to 54.5 ng/m3 (geometric mean, 8.1 ng/m3). TWD was a significant predictor of natural log personal air Mn concentration (lnMn) with an associated decrease of 0.075 lnMn for each km TWD ($p<0.05$, 95% CI −0.13 to −0.01). Personal Mn exposures were positively associated with stationary air Mn levels and inversely associated with wind speed. A child's location (home and school) relative to the refinery is a significant predictor of personal Mn exposure. Wind speed is also an important contributor to personal Mn exposure.

Zoni et al. (2013). OLFACTORY FUNCTIONS AT THE INTERSECTION BETWEEN ENVIRONMENTAL EXPOSURE TO MANGANESE AND PARKINSONISM *J Trace Elem Med Biol. 2012 June ; 26(2-3): 179–182. doi:10.1016/j.jtemb.2012.04.023.*

The olfactory function can be affected by occupational and environmental exposure to various neurotoxicants that can be transported through the olfactory pathway. Olfactory impairment is a highly recurrent non-motor dysfunction in Parkinson's disease and is considered an early predictive sign of neurodegeneration. Changes in olfactory perception may be caused by a dopaminergic dysregulation, possibly related to changes at the level of dopamine receptors.

Manganese is an essential element that can become neurotoxic in various conditions inducing an overload in the organism. Being actively transported through the olfactory tract, manganese can cause impairment of olfactory function and motor coordination in different age groups like children and elderly. Odor and motor changes are interrelated and may be caused by a Mn-induced dopaminergic dysregulation affecting both functions. Given these findings, further

research is imperative on the possible role of manganese exposure as a pathogenetic factor for Parkinsonism.

Lucchini et al (2013) **TREMOR, OLFACTORY AND MOTOR CHANGES IN ITALIAN ADOLESCENTS EXPOSED TO HISTORICAL FERROMANGANESE EMISSION**

Background and Objective—Increased prevalence of Parkinsonism was observed in Valcamonica, Italy, a region impacted by ferroalloy plants emissions containing manganese and other metals for a century until 2001. The aim of this study was to assess neurobehavioral functions in adolescents from the impacted region and the reference area of Garda Lake.
Methods—Adolescents age 11–14 yrs were recruited through the school system for neurobehavioral testing. Metals including manganese, lead, iron, zinc, copper were measured in airborne particulate matter collected with 24-hour personal samplers, and in soil, tap water, blood, urine and hair. Independent variables included parental education and socio-economic status, children's body mass index, number of siblings, parity order, smoking and drinking habits.
Results—A total of 311 subjects (49.2% females), residing in either the exposed (n=154) or the reference (n=157) area participated. Average airborne and soil manganese were respectively 49.5 ng/m3 (median 31.4, range 1.24–517) and 958 ppm (median 897, range 465–1729) in the impacted area, and 27.4 ng/m3 (median 24.7, range 5.3–85.9) ng/m3 and 427 ppm (median 409 range 160–734) in the reference area. Regression models showed significant impairment of motor coordination (Luria-Nebraska test, $p=0.0005$), hand dexterity (Aiming Pursuit test, $p=0.0115$) and odor identification (Sniffin' task, $p=0.003$) associated with soil manganese. Tremor intensity was positively associated with blood ($p=0.005$) and hair ($p=0.01$) manganese.
Conclusion—Historical environmental exposure to manganese from ferroalloy emission reflected by the concentration in soil and the biomarkers was associated with subclinical deficits in olfactory and motor function among adolescents.

Racette et al (2013) **Pathophysiology of manganese-associated neurotoxicity.**

Conference Summary

Manganese (Mn) is a well established neurotoxin associated with specific damage to the basal ganglia in humans. The phenotype associated with Mn neurotoxicity was first described in two workers with occupational exposure to Mn oxide.(Couper, 1837) Although the description did not use modern clinical terminology, a parkinsonian illness characterized by slowness of movement (bradykinesia), masked facies, and gait impairment (postural instability) appears to have predominated. Nearly 100 years later an outbreak of an atypical parkinsonian illness in a Chilean Mn mine provided a phenotypic description of a fulminant neurologic disorder with parkinsonism, dystonia, and neuropsychiatric symptoms.(Rodier J, 1955) Exposures associated with this syndrome were massive and an order of magnitude greater than modern exposures.(Rodier J, 1955; Hobson et al., 2011) The clinical syndrome associated with Mn neurotoxicity has been called manganism.

Modern exposures to Mn occur primarily through occupations in the steel industry and welding. These exposures are often chronic and varied, occurring over decades in the healthy workforce.

Although the severe neurologic disorder described by Rodier and Couper are no longer seen, several reports have suggested a possible increased risk of neurotoxicity in these workers.(Racette et al., 2005b; Bowler et al., 2007; Harris et al., 2011) Based upon limited prior imaging and pathologic investigations into the pathophysiology of neurotoxicity in Mn exposed workers, (Huang et al., 2003) many investigators have concluded that the syndrome spares the dopamine system distinguishing manganism from Parkinson disease (PD), the most common cause of parkinsonism in the general population, and a disease with characteristic degenerative changes in the dopaminergic system.(Jankovic, 2005)

The purpose of this symposium was to highlight recent advances in the understanding of the pathophysiology of Mn associated neurotoxicity from C. elegans to humans. Dr. Aschner's presentation discussed mechanisms of dopaminergic neuronal toxicity in C. elegans and demonstrates a compelling potential role of Mn in dopaminergic degeneration. Dr. Guilarte's experimental, non-human primate model of Mn neurotoxicity suggests that Mn decreases dopamine release in the brain without loss of neuronal integrity markers, including dopamine. Dr. Racette's presentation demonstrates a unique pattern of dopaminergic dysfunction in active welders with chronic exposure to Mn containing welding fumes. Finally, Dr. Dydak presented novel magnetic resonance (MR) spectroscopy data in Mn exposed smelter workers and demonstrated abnormalities in the thalamus and frontal cortex for those workers. This symposium provided some converging evidence of the potential neurotoxic impact of Mn on the dopaminergic system and challenged existing paradigms on the pathophysiology of Mn in the central nervous system.

████████████████████████████ **"Early life versus lifelong oral manganese exposure differently impairs skilled forelimb performance in adult rats."** Neurotoxicol Teratol **38C**: 36-45. [HYPERLINK "http://www.ncbi.nlm.nih.gov/pubmed/23623961"].

Abstract: Recent studies of children suggest that exposure to elevated manganese (Mn) levels disrupts aspects of motor, cognitive and behavioral functions that are dependent on dopamine brain systems. Although basal ganglia motor functions are well-known targets of adult occupational Mn exposure, the extent of motor function deficits in adults as a result of early life Mn exposure is unknown. Here we used a rodent model early life versus lifelong oral Mn exposure and the Montoya staircase test to determine whether developmental Mn exposure produces long-lasting deficits in sensorimotor performance in adulthood. Long-Evans male neonate rats (n=11/treatment) were exposed daily to oral Mn at levels of 0, 25, or 50mg Mn/kg/d from postnatal day (PND) 1-21 (early life only), or from PND 1-throughout life. Staircase testing began at age PND 120 and lasted 1month to objectively quantify measures of skilled forelimb use in reaching and pellet grasping/retrieval performance. Behavioral reactivity also was rated on each trial. Results revealed that (1) behavioral reactivity scores were significantly greater in the Mn-exposed groups, compared to controls, during the staircase acclimation/training stage, but not the latter testing stages, (2) early life Mn exposure alone caused long-lasting impairments in fine motor control of reaching skills at the higher, but not lower Mn dose, (3) lifelong Mn exposure from drinking water led to widespread impairment in reaching and grasping/retrieval performance in adult rats, with the lower Mn dose group showing the greatest impairment, and (4) lifelong Mn exposure produced similar (higher Mn group) or more severe (lower Mn group) impairments compared to their early life-only Mn exposed counterparts. Collectively, these

results substantiate the emerging clinical evidence in children showing associations between environmental Mn exposure and deficits in fine sensorimotor function. They also show that the objective quantification of skilled motor performance using the staircase test can serve as a sensitive measure of early life insults from environmental agents.

"Manganese-exposed developing rats display motor deficits and striatal oxidative stress that are reversed by Trolox." <u>Arch Toxicol</u> 87(7): 1231-1244. http://www.ncbi.nlm.nih.gov/pubmed/23385959

Abstract: While manganese (Mn) is essential for proper central nervous system (CNS) development, excessive Mn exposure may lead to neurotoxicity. Mn preferentially accumulates in the basal ganglia, and in adults it may cause Parkinson's disease-like disorder. Compared to adults, younger individuals accumulate greater Mn levels in the CNS and are more vulnerable to its toxicity. Moreover, the mechanisms mediating developmental Mn-induced neurotoxicity are not completely understood. The present study investigated the developmental neurotoxicity elicited by Mn exposure (5, 10 and 20 mg/kg; i.p.) from postnatal day 8 to PN27 in rats. Neurochemical analyses were carried out on PN29, with a particular focus on striatal alterations in intracellular signaling pathways (MAPKs, Akt and DARPP-32), oxidative stress generation and cell death. Motor alterations were evaluated later in life at 3, 4 or 5 weeks of age. Mn exposure (20 mg/kg) increased p38(MAPK) and Akt phosphorylation, but decreased DARPP-32-Thr-34 phosphorylation. Mn (10 and 20 mg/kg) increased caspase activity and F2-isoprostane production (a biological marker of lipid peroxidation). Paralleling the changes in striatal biochemical parameters, Mn (20 mg/kg) also caused motor impairment, evidenced by increased falling latency in the rotarod test, decreased distance traveled and motor speed in the open-field test. Notably, the antioxidant Trolox reversed the Mn (20 mg/kg)-dependent augmentation in p38(MAPK) phosphorylation and reduced the Mn (20 mg/kg)-induced caspase activity and F2-isoprostane production. Trolox also reversed the Mn-induced motor coordination deficits. These findings are the first to show that long-term exposure to Mn during a critical period of neurodevelopment causes motor coordination dysfunction with parallel increment in oxidative stress markers, p38(MAPK) phosphorylation and caspase activity in the striatum. Moreover, we establish Trolox as a potential neuroprotective agent given its efficacy in reversing the Mn-induced neurodevelopmental effects.

Manganese exposure and cognitive deficits: A growing concern for manganese neurotoxicity. Neurotoxicology (2012), http://dx.doi.org/10.1016/j.neuro.2012.03.009

Abstract: This symposium comprised five oral presentations dealing with recent findings on Mn-related cognitive and motor changes from epidemiological studies across the life span. The first contribution highlighted the usefulness of functional neuroimaging of the central nervous system (CNS) to evaluate cognitive as well as motor deficits in Mn-exposed welders. The second dealt with results of two prospective studies in Mn-exposed workers or welders showing that after decrease of Mn exposure the outcome of reversibility in adverse CNS effects may differ for motor and cognitive function and, in addition the issue of plasma Mn as a reliable biomarker for Mn exposure in welders has been addressed. The third presentation showed a brief overview of the results of an ongoing study assessing the relationship between environmental airborne Mn

exposure and neurological or neuropsychological effects in adult Ohio residents living near a Mn point source. The fourth paper focused on the association between blood Mn and neurodevelopment in early childhood which seems to be sensitive to both low and high Mn concentrations. The fifth contribution gave an overview of six studies indicating a negative impact of excess environmental Mn exposure from air and drinking water on children's cognitive performance, with special attention to hair Mn as a potential biomarker of exposure. These studies highlight a series of questions about Mn neurotoxicity with respect to cognitive processes, forms and routes of exposure, adequate biomarkers of exposure, gender differences, susceptibility and exposure limits with regard to age.

ATSDR (2012). **Draft Toxicological Profile for Manganese.** U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry.

Bowman, et al (2012). Role of manganese in neurodegenerative diseases. J Trace Elem Med Biol. 2011 December ; 25(4): 191–203. doi:10.1016/j.jtemb.2011.08.144.

Mn is an essential ubiquitous trace element required for normal growth, development and cellular homeostasis [1]. Specifically, Mn is important in bone formation, fat and carbohydrate metabolism, blood sugar regulation, and calcium absorption. In humans and animals, Mn functions as a required cofactor of several enzymes necessary for neuronal and glial cell function, as well as enzymes involved in neurotransmitter synthesis and metabolism [2, 3, 4]. Furthermore, *in vitro* data has implicated Mn in the induction of stellate process formation by astrocytes [5]. Mn exists in various chemical forms including oxidation states ($Mn2+$, $Mn3+$, $Mn4+$ $Mn6+$, $Mn7+$), salts (sulfate and gluconate), and chelates (aspartate, fumarate, succinate). The versatile chemical properties of Mn have enabled its industrial usage in making glass and ceramics, adhesives, welding, paint, gasoline anti-knock additives (methylcyclopentadienyl manganese tricarbonyl (MMT), and many others. While uncommon, Mn deficiency can contribute to birth defects, impaired fertility, bone malformation, weakness, and enhanced susceptibility to seizures [6, 7]. The routes of Mn exposure are mainly through dietary intake, dermal absorption, and inhalation.

Moreover, Mn in the diet is found mostly in whole grains, nuts, and seeds, tea, legumes, pineapple, and beans. Despite its essential role in multiple metabolic functions, excessive Mn exposure can accumulate in the brain and has been associated with dysfunction of the basal ganglia system that causes a severe neurological disorder similar to PD [8].

1. Mn essentiality and toxicity
1b. Mn transporters in the brain
1c. Methods for detecting Mn in biological specimens
2. An overview of the role for Mn and other metals in neurodegeneration
3. Manganese exposure and Parkinson's disease
3a. Mangansim vs. PD
3b. Human exposure to Mn and relationship to PD
3c. α-synuclein and Mn-related protein aggregation
3d. Mitochondrial dysfunction, Mn and PD

4. Mn and HD

4a. A role for environmental factors in HD

4b. Links between HTT function and metals

4c. A role for altered metal homeostasis and toxicity in HD neuropathology

4d. Discovery of a disease-toxicant interaction between HD and Mn exposure

5. Mn and Amyotrophic Lateral Sclerosis (ALS)

6. Mn and prion diseases

7. Mn and Alzheimer's disease (AD)

8. Future directions

Molina et al (2012). Ingestion of Mn and Pb by rats during and after pregnancy alters iron metabolism and behavior in offspring. *NeuroToxicology* **2011**, *32* (4), 413-422.

Manganese (Mn) and lead (Pb) exposures during developmental period can impair development by direct neurotoxicity or through interaction with iron metabolism. Therefore, we examined the effects of maternal ingestion of Mn or Pb in drinking water during gestation and lactation on iron metabolism as well as behavior in their offspring. Pregnant dams were given distilled water, 4.79 mg/ml Mn, or 2.84 mg/ml Pb in drinking water during gestation and lactation. Pups were studied at time of weaning for 59Fe absorption from the gut, duodenal Divalent Metal Transporter 1 (DMT1) expression, hematological parameters, and anxiety-related behavior using an Elevated Plus Maze (EPM) test. Metal-exposed pups had lower body weights and elevated blood and brain concentrations of the respective metal. Pb-exposed pups had lower hematocrits and higher blood Zn protoporphyrin levels. In contrast, Mn exposed pups had normal hematological parameters but significantly reduced Zn protoporphyrin. Pharmacokinetic studies using 59Fe showed that intestinal absorption in metal-exposed pups was not different from controls, nor was it correlated with duodenal DMT1 expression. However, intravenously injected 59Fe was cleared more slowly in Pb-exposed pups resulting in higher plasma levels. The overall tissue uptake of 59Fe was lower
in Mn-exposed and lower in the brain in Pb-exposed pups. The EPM test demonstrated that Mnexposed, but not Pb-exposed, pups had lower anxiety-related behavior compared to controls. We conclude that gestational and lactational exposures to Mn or Pb differentially alter Fe metabolism and anxiety-related behavior. The data suggest that perturbation in Fe metabolism may contribute to the pathophysiologic consequences of Mn and Pb exposure during early development.

Ding et al (2012) **Manganese is toxic to spiral ganglion neurons and hair cells in vitro.** Neurotoxicology. 2011 March ; 32(2): 233–241. doi:10.1016/j.neuro.2010.12.003.

Occupational exposure to high atmospheric levels of Mn produces a severe and debilitating disorder known as manganism characterized by extrapyramidal disturbances similar to that seen in Parkinson's disease. Epidemiological and case studies suggest that persistent exposures to Mn may have deleterious effects on other organs including the auditory system and hearing. Mn accumulates in the inner ear following acute exposure raising the possibility that it can damage the sensory hair cells that convert sound into neural activity or spiral ganglion neurons (SGN) that transmit acoustic information from the hair cells to the brain via the auditory nerve. In this paper we demonstrate for first time that Mn causes significant damage to the sensory hair cells, peripheral auditory nerve fibers (ANF) and SGN in cochlear organotypic cultures isolated from

postnatal day three rats. The peripheral ANF that make synaptic contact with the sensory hair cells were particularly vulnerable to Mn toxicity; damage occurred at concentrations as low 0.01 mM and increased with dose and duration of Mn exposure. Sensory hair cells, in contrast, were slightly more resistant to Mn toxicity than the ANF. Mn induced an atypical pattern of sensory cell damage; Mn was more toxic to inner hair cells (IHC) than outer hair cells (OHC) and in addition, IHC loss was relatively uniform along the length of the cochlea. Mn also caused significant loss and shrinkage of SGN soma. These findings are the first to demonstrate that Mn can produce severe lesions to both neurons and hair cells in the postnatal inner ear.

Bagga, P., Patel, A. B. (2012) **Regional cerebral metabolism in mouse under chronic manganese exposure: Implications for Manganism**. *Neurochemistry International* **2012**, *60* (2), 177-185

Chronic manganese (Mn) exposure in rodents, non-human primates and humans has been linked to Parkinson's disease like condition known as Manganism. Mn being a cofactor for many enzymes in brain has been known to be accumulated in various regions differentially and thus exert toxic effect upon chronic overexposure. In present study, neuropathology of Manganism was investigated by evaluating regional neuronal and astroglial metabolism in mice under chronic Mn exposure. Male C57BL6 mice were treated with MnCl2 (25 mg/kg, i.p.) for 21 days. Cerebral metabolism was studied by co-infusing [U-13C6]glucose and [2-13C]acetate, and monitoring 13C labeling of amino acids in brain tissue extract using 1H–[13C] and 13C–[1H]-NMR spectroscopy. Glutamate, choline, N-acetyl aspartate and myo-inositol were found to be reduced in thalamus and hypothalamus indicating a loss in neuronal and astroglial cells due to Mn neurotoxicity.

Reduced labeling of GluC4 from [U-13C6]glucose and [2-13C]acetate indicates an impairment of glucose oxidation by glutamatergic neurons and glutamate–glutamine neurotransmitter cycle in cortex, striatum, thalamus–hypothalamus and olfactory bulb with chronic Mn exposure. Additionally, reduced labeling of GlnC4 from [2-13C]acetate indicates a decrease in acetate oxidation by astroglia in the same regions. However, GABAergic function was alleviated only in thalamus–hypothalamus. Our findings indicate that chronic Mn impairs excitatory (glutamatergic) function in the majority of regions of brain while inhibitory (GABAergic) activity is perturbed only in basal ganglia.

ATSDR Public Health Statement: Manganese (Sept 2012).

Fordahl, S., P. Cooney, et al. (2012) **"Waterborne manganese exposure alters plasma, brain, and liver metabolites accompanied by changes in stereotypic behaviors."** Neurotoxicology and Teratology **34**(1): 27-36. http://www.ncbi.nlm.nih.gov/pubmed/22056924.

Abstract: Overexposure to waterborne manganese (Mn) is linked with cognitive impairment in children and neurochemical abnormalities in other experimental models. In order to characterize the threshold between Mn-exposure and altered neurochemistry, it is important to identify biomarkers that positively correspond with brain Mn-accumulation. The objective of this study was to identify Mn-induced alterations in plasma, liver, and brain metabolites using liquid/gas chromatography–time of flight–mass spectrometry metabolomic analyses; and to monitor

corresponding Mn-induced behavior changes. Weanling Sprague–Dawley rats had access to deionized drinking water either Mn-free or containing 1 g Mn/L for 6 weeks. Behaviors were monitored during the sixth week for a continuous 24 h period while in a home cage environment using video surveillance. Mn-exposure significantly increased liver, plasma, and brain Mn concentrations compared to control, specifically targeting the globus pallidus (GP). Mn significantly altered 98 metabolites in the brain, liver, and plasma; notably shifting cholesterol and fatty acid metabolism in the brain (increased oleic and palmitic acid; 12.57 and 15.48 fold change (FC), respectively), and liver (increased oleic acid, 14.51 FC; decreased hydroxybutyric acid, − 14.29 FC). Additionally, Mn-altered plasma metabolites homogentisic acid, chenodeoxycholic acid, and aspartic acid correlated significantly with GP and striatal Mn. Total distance traveled was significantly increased and positively correlated with Mn-exposure, while nocturnal stereotypic and exploratory behaviors were reduced with Mn-exposure and performed largely during the light cycle compared to unexposed rats. These data provide putative biomarkers for Mn-neurotoxicity and suggest that Mn disrupts the circadian cycle in rats.

Khan, K., G. A. Wasserman, et al. (2012) **"Manganese exposure from drinking water and children's academic achievement."** Neurotoxicology 33(1): 91-97. [HYPERLINK "http://www.ncbi.nlm.nih.gov/pubmed/22182530"].

Abstract: Drinking water manganese (WMn) is a potential threat to children's health due to its associations with a wide range of outcomes including cognitive, behavioral and neuropsychological effects. Although adverse effects of Mn on cognitive function of the children indicate possible impact on their academic achievement little evidence on this issue is available. Moreover, little is known regarding potential interactions between exposure to Mn and other metals, especially water arsenic (WAs). In Araihazar, a rural area of Bangladesh, we conducted a cross-sectional study of 840 children to investigate associations between WMn and WAs and academic achievement in mathematics and languages among elementary school-children, aged 8-11 years. Data on As and Mn exposure were collected from the participants at the baseline of an ongoing longitudinal study of school-based educational intervention. Annual scores of the study children in languages (Bangla and English) and mathematics were obtained from the academic achievement records of the elementary schools. WMn above the WHO standard of 400mug/L was associated with 6.4% score loss (95% CI=-12.3 to -0.5) in mathematics achievement test scores, adjusted for WAs and other sociodemographic variables. We did not find any statistically significant associations between WMn and academic achievement in either language. Neither WAs nor urinary As was significantly related to any of the three academic achievement scores. Our finding suggests that a large number of children in rural Bangladesh may experience deficits in mathematics due to high concentrations of Mn exposure in drinking water.

Bouchard, M. F., S. Sauve, et al. (2011) **Intellectual Impairment in School-Age Children Exposed to Manganese from Drinking Water.** Environ Health Perspect 119: 138-143. [HYPERLINK "http://www.ncbi.nlm.nih.gov/pubmed/20855239"]

Abstract: Background: Manganese is an essential nutrient, but in excess, can be a potent neurotoxicant. Despite the common occurrence of manganese in groundwater, the risks associated with this source of exposure are largely unknown. Objectives: Our first aim was to

assess the relations between exposure to manganese from drinking water and children's intellectual quotient (IQ). Secondly, we examined the relations between manganese exposures from water consumption and from the diet with children's hair manganese concentration. Methods: This cross-sectional study included 362 children ages 6 to 13 years living in communities supplied by groundwater. Manganese concentration was measured in home tap water (MnW) and children's hair (MnH). We estimated manganese intake from water ingestion and the diet using a food frequency questionnaire, and assessed IQ with the Wechsler Abbreviated Scale of Intelligence. Results: The median MnW in children's home tap water was 34 microg/L (range: 1-2700 microg/L). MnH increased with manganese intake from water consumption, but not with dietary manganese intake. Higher MnW and MnH were significantly associated with lower IQ scores. A 10-fold increase in MnW was associated with a decrease of 2.4 IQ points (95% confidence intervals: -3.9, -0.9; P < 0.01), adjusting for maternal intelligence, family income, and other potential confounders. There was a 6.2-IQ point difference between children in the lowest and highest MnW quintiles. MnW was more strongly associated with Performance IQ than Verbal IQ. Conclusions: The findings of this cross-sectional study suggest that exposure to manganese at levels common in groundwater is associated with intellectual impairment in children.

Hong Chen, Ray Copes (2011) **Manganese in Drinking Water and Intellectual Impairment in School-Age Children** volume 119 | number 6 | June 2011 • Environmental Health Perspectives*doi:10.1289/ehp.1103485*

We read with interest the the article by Bouchard et al. (2011) on the effect of manganese in drinking water on children's IQ (intelligence quotient). In this cross-sectional study, the authors examined IQ scores in relation to manganese exposure using four exposure metrics: *a*) concentration of manganese in tap water; *b*) concentration of manganese in hair samples; *c*) estimate of manganese intake from water consumption; and *e*) estimate of manganese intake from diet consumption.

Manganese in Drinking Water: Bouchard Responds *doi:10.1289/ehp.1103485R*

Chen and Copes raise some interesting issues regarding our article (Bouchard et al. 2011). In our study we investigated the change in IQ scores with respect to different exposure metrics for manganese. One of these metrics was home tap water manganese concentration, which was strongly associated with IQ deficits. Chen and Copes indicate that they consider it inappropriate to include in this analysis children who did not drink tap water at home. Second, they note that even for children in the highest quintile of water manganese concentration, the intake of manganese from water ingestion is below the recommended dietary manganese intake (Institute of Medicine 2001). In response…

Khan et al (2011) **Manganese Exposure from Drinking Water and Children's Academic Achievement** *Neurotoxicology. 2012 January ; 33(1): 91–97. doi:10.1016/j.neuro.2011.12.002.*

Drinking water manganese (WMn) is a potential threat to children's health due to its associations with a wide range of outcomes including cognitive, behavioral and neuropsychological effects.

Although adverse effects of Mn on cognitive function of the children indicate possible impact on their academic achievement little evidence on this issue is available.. Moreover, little is known regarding potential interactions between exposure to Mn and other metals, especially water arsenic (WAs). In Araihazar, a rural area of Bangladesh, we conducted a cross-sectional study of 840 children to investigate associations between WMn and WAs and academic achievement in mathematics and languages among elementary school-children, aged 8–11 years. Data on As and Mn exposure were collected from the participants at the baseline of an ongoing longitudinal study of school-based educational intervention. Annual scores of the study children in languages (Bangla and English) and mathematics were obtained from the academic achievement records of the elementary schools. WMn above the WHO standard of 400 µg/L was associated with 6.4 percentage score loss (95% CI=0.5, 12.3) in mathematics achievement test scores, adjusted for WAs and other sociodemographic variables. We did not find any significant associations between WMn and academic achievement in either language. Neither WAs nor urinary As was significantly related to any of the three academic achievement scores. Our finding suggests that a large number of children in rural Bangladesh may experience deficits in mathematics due to high concentrations of Mn exposure in drinking water.

Khan, K., P. Factor-Litvak, et al. (2011). **Manganese Exposure from Drinking Water and Children's Classroom Behavior in Bangladesh.** Environmental Health Perspectives **119**(10): 1501-1506. http://www.ncbi.nlm.nih.gov/pubmed/21493178

Abstract: Background: Evidence of neurological, cognitive, and neuropsychological effects of manganese (Mn) exposure from drinking water (WMn) in children has generated widespread public health concern. At elevated exposures, Mn has been associated with increased levels of externalizing behaviors,including irritability, aggression, and impulsivity. Little is known about potential effects at lower exposures, especially in children. Moreover, little is known regarding potential interactions between exposure to Mn and other metals, especially arsenic (As).
Objectives: We conducted a cross-sectional study of 201 children to investigate associations of Mn and As in tube well water with classroom behavior among elementary school children, 8–11 years of age, in Araihazar, Bangladesh.
Methods: Data on exposures and behavioral outcomes were collected from the participants at the baseline of an ongoing longitudinal study of child intelligence. Study children were rated by their school teachers on externalizing and internalizing items of classroom behavior using the standardized Child Behavior Checklist-Teacher's Report Form (CBCL-TRF).
Results: Log-transformed WMn was positively and significantly associated with TRF internalizing [estimated β = 0.82; 95% confidence interval (CI), 0.08–1.56; p = 0.03], TRF externalizing (estimated β = 2.59; 95% CI, 0.81–4.37; p =0.004), and TRF total scores (estimated β = 3.35; 95% CI, 0.86–5.83; p = 0.008) in models that adjusted for log-transformed water arsenic (WAs) and sociodemographic covariates. We also observed a positive monotonic dose–response relationship between WMn and TRF externalizing and TRF total scores among the participants of the study. We did not find any significant associations between WAs and various scales of TRF scores.
Conclusion: These observations reinforce the growing concern regarding the neurotoxicologic effects of WMn in children.

Arsenic Exposure and Motor Function among Children in Bangladesh. Environmental Health Perspectives **119**(11): 1665-1670.
http://www.ncbi.nlm.nih.gov/pubmed/21742576

Abstract: Background: Several reports indicate that drinking water arsenic (WAs) and manganese (WMn) are associated with children's intellectual function. Very little is known, however, about possible associations with other neurologic outcomes such as motor function.
Methods: We investigated the associations of WAs and WMn with motor function in 304 children in Bangladesh, 8-11 years of age. We measured As and Mn concentrations in drinking water, blood,urine, and toenails. We assessed motor function with the Bruininks-Oseretsky test, version 2, in four subscales-fine manual control (FMC), manual coordination (MC), body coordination (BC), and strength and agility-which can be summarized with a total motor composite score (TMC).
Results: Log-transformed blood As was associated with decreases in TMC [$\beta = -3.63$; 95% confidence interval (CI): -6.72, -0.54; $p < 0.01$], FMC ($\beta = -1.68$; 95% CI: -3.19, -0.18; $p < 0.05$), and BC ($\beta = -1.61$; 95% CI: -2.72, -0.51; $p < 0.01$), with adjustment for sex, school attendance, head circumference, mother's intelligence, plasma ferritin, and blood Mn, lead, and selenium. Other measures of As exposure (WAs, urinary As, and toenail As) also were inversely associated with motor function scores, particularly TMC and BC. Square-transformed blood selenium was positively associated with TMC ($\beta = 3.54$; 95% CI: 1.10, 6.0; $p < 0.01$), FMC ($\beta = 1.55$; 95% CI: 0.40, 2.70; $p < 0.005$), and MC ($\beta = 1.57$; 95% CI: 0.60, 2.75; $p < 0.005$) in the unadjusted models. Mn exposure was not significantly associated with motor function.
Conclusion: Our research demonstrates an adverse association of As exposure and a protective association of Se on motor function in children.

Arsenic and manganese exposure and children's intellectual function. Neurotoxicology **32**(4): 450-457.
http://www.ncbi.nlm.nih.gov/pubmed/21453724

Abstract: Recently, epidemiologic studies of developmental neurotoxicology have been challenged to increase focus on co-exposure to multiple toxicants. Earlier reports, including our own work in Bangladesh, have demonstrated independent associations between neurobehavioral function and exposure to both arsenic (As) and manganese (Mn) in school-aged children. Our earlier studies, however, were not designed to examine possible interactive effects of exposure to both As and Mn. To allow investigation of possible synergistic impact of simultaneous exposures, we recruited a new sample of 299 8-11 year old children, stratified by design on As (above and below 10mug/L) and Mn (above and below 500mug/L) concentrations of household wells. When adjusted only for each other, both As and Mn in whole blood (BAs; BMn) were significantly negatively related to most WISC-IV subscale scores. With further adjustment for socio-demographic features and ferritin, BMn remained significantly associated with reduced Perceptual Reasoning and Working Memory scores; associations for BAs, and for other subscales, were expectably negative, significantly for Verbal Comprehension. Urinary As (per gram creatinine) was significantly negatively associated with Verbal Comprehension scores, even with adjustment for BMn and other contributors. Mn by As interactions were not significant in adjusted or unadjusted models (all p's>0.25). Findings are consistent with other reports

documenting adverse impact of both As and Mn exposure on child developmental outcomes, although associations appear muted at these relatively low exposure levels.

Background: Several reports indicate that drinking water arsenic (WAs) and manganese (WMn) are associated with children's intellectual function. Very little is known, however, about possible associations with other neurologic outcomes such as motor function.
Methods: We investigated the associations of WAs and WMn with motor function in 304 children in Bangladesh, 8–11 years of age. We measured As and Mn concentrations in drinking water, blood, urine, and toenails. We assessed motor function with the Bruininks-Oseretsky test, version 2, in four subscales—fine manual control (FMC), manual coordination (MC), body coordination (BC), and strength and agility—which can be summarized with a total motor composite score (TMC).
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Conclusion: Our research demonstrates an adverse association of As exposure and a protective association of Se on motor function in children.

The present study examines the effects of divalent and trivalent Manganese (Mn2+/Mn3+) mixture inhalation on mice to obtain a novel animal model of Parkinson disease (PD) inducing bilateral and progressive dopaminergic cell death, correlate those alterations with motor disturbances, and determine whether l-DOPA treatment improves the behavior, to ensure that the alterations are of dopaminergic origin. CD-1 male mice inhaled a mixture of Manganese chloride and Manganese acetate, one hour twice a week for five months. BeforeMn exposure, animals were trained to perform motor function tests and were evaluated each week after the exposure.

By the end of Mn exposure, 10 mice were orally treated with 7.5mg/kg l-DOPA. After 5 months of Mn mixture inhalation, striatal dopamine content decreased 71%, the SNc showed important reduction in the number of TH-immunopositive neurons, mice developed akinesia, postural instability, and action tremor; these motor alterations were reverted with l-DOPA treatment. Our data provide evidence thatMn2+/Mn3+ mixture inhalation produces similar morphological,

neurochemical, and behavioral alterations to those observed in PD providing a useful experimental model for the study of this neurodegenerative disease.

Contamination of the environment by metals is recognized as a threat to health. One of their targets is the brain, and the adverse functional effects they induce are reflected by neurobehavioral assessments. Lead, manganese, and methylmercury are the metal contaminants linked most comprehensively to such disorders. Because many of these adverse effects can appear later in life, clues to the role of metals as risk factors for neurodegenerative disorders should be sought in the exposure histories of aging populations. A review of the available literature offers evidence that all three metals can produce, in advanced age, manifestations of neurobehavioral dysfunction associated with neurodegenerative disease. Among the critical unresolved questions is timing; that is, during which periods of the lifespan, including early development, do environmental exposures lay the foundations for their ultimate effects?

Abstract: Manganese (Mn) is an essential element for humans, animals, and plants and is required for growth, development, and maintenance of health. Mn is present in most tissues of all living organisms and is present naturally in rocks, soil, water, and food. High-dose oral, parenteral, or inhalation exposures are associated with increased tissue Mn levels that may lead to development of adverse neurological, reproductive, or respiratory effects. Manganese-induced clinical neurotoxicity is associated with a motor dysfunction syndrome commonly referred to as manganism. Because Mn is an essential element and absorption and excretion are homeostatically regulated, a reasonable hypothesis is that there should be no adverse effects at low exposures. Therefore, there should be a threshold for exposure, below which adverse effects may occur only rarely, if at all, and the frequency of occurrence of adverse effects may increase with higher exposures above that threshold. Lowest-observed-adverse-effect levels (LOAELs), no-observed-adverse-effect levels (NOAELs), and benchmark dose levels (BMDs) have been derived from studies that were conducted to evaluate subclinical neurotoxicity in human occupational cohorts exposed to Mn. Although there is some uncertainty about the predictive value of the subclinical neuromotor or neurobehavioral effects that were observed in these occupational cohort studies, results of the neurological tests were used in risk assessments to establish guidelines and regulations for ambient air levels of Mn in the environment. A discussion of the uncertainties associated with these tests is provided in this review. The application of safety and uncertainty factors result in guidelines for ambient air levels that are lower than the LOAELs, NOAELs, or BMDs from occupational exposure studies by an order of magnitude, or more. Specific early biomarkers of effect, such as subclinical neurobehavioral or neurological changes or magnetic resonance imaging (MRI) changes, have not been established or validated for Mn, although some studies attempted to correlate certain biomarkers with neurological effects. Pharmacokinetic studies with rodents and monkeys provide valuable

information about the absorption, bioavailability, and tissue distribution of various Mn compounds with different solubilities and oxidation states in different age groups. These pharmacokinetic studies showed that rodents and primates maintain stable tissue Mn levels as a result of homeostatic mechanisms that tightly regulate absorption and excretion of ingested Mn and limit tissue uptake at low to moderate levels of inhalation exposure. In addition, physiologically based pharmacokinetic (PBPK) models are being developed to provide for the ability to conduct route-to-route extrapolations, evaluate nasal uptake to the central nervous system (CNS), and determine life-stage differences in Mn pharmacokinetics. Such models will facilitate more rigorous quantitative analysis of the available human pharmacokinetic data for Mn and will be used to identify situations that may lead to increased brain accumulation related to altered Mn kinetics in different human populations, and to develop quantitatively accurate predictions of elevated Mn levels that may serve as a basis of dosimetry-based risk assessments. Such dosimetry-based risk assessments will permit for the development of more scientifically refined and robust recommendations, guidelines, and regulations for Mn levels in the ambient environment and occupational settings.

Boyes, W. K. (2010) **"Essentiality, toxicity, and uncertainty in the risk assessment of manganese."** J Toxicol Environ Health A **73**(2): 159-165. [HYPERLINK "http://www.ncbi.nlm.nih.gov/pubmed/20077286"].

Abstract: Risk assessments of manganese by inhalation or oral routes of exposure typically acknowledge the duality of manganese as an essential element at low doses and a toxic metal at high doses. Previously, however, risk assessors were unable to describe manganese pharmacokinetics quantitatively across dose levels and routes of exposure, to account for mass balance, and to incorporate this information into a quantitative risk assessment. In addition, the prior risk assessment of inhaled manganese conducted by the U.S. Environmental Protection Agency (EPA) identified a number of specific factors that contributed to uncertainty in the risk assessment. In response to a petition regarding the use of a fuel additive containing manganese, methylcyclopentadienyl manganese tricarbonyl (MMT), the U.S. EPA developed a test rule under the U.S. Clean Air Act that required, among other things, the generation of pharmacokinetic information. This information was intended not only to aid in the design of health outcome studies, but also to help address uncertainties in the risk assessment of manganese. To date, the work conducted in response to the test rule has yielded substantial pharmacokinetic data. This information will enable the generation of physiologically based pharmacokinetic (PBPK) models capable of making quantitative predictions of tissue manganese concentrations following inhalation and oral exposure, across dose levels, and accounting for factors such as duration of exposure, different species of manganese, and changes of age, gender, and reproductive status. The work accomplished in response to the test rule, in combination with other scientific evidence, will enable future manganese risk assessments to consider tissue dosimetry more comprehensively than was previously possible.

Claus Henn, B., A. S. Ettinger, et al. (2010). **"Early postnatal blood manganese levels and children's neurodevelopment."** Epidemiology **21**(4): 433-439.
http://www.ncbi.nlm.nih.gov/pubmed/20549838.

Abstract: Recent evidence suggests that low-level environmental exposure to manganese adversely affects child growth and neurodevelopment. Previous studies have addressed the effects of prenatal exposure, but little is known about developmental effects of early postnatal exposure.
METHODS: We studied 448 children born in Mexico City from 1997 through 2000, using a longitudinal study to investigate neurotoxic effects of early-life manganese exposure. Archived blood samples, collected from children at 12 and 24 months of age, were analyzed for manganese levels using inductively coupled plasma mass spectrometry. Mental and psychomotor development were scored using Bayley Scales of Infant Development at 6-month intervals between 12 and 36 months of age.
RESULTS: At 12 months of age, the mean (SD) blood manganese level was 24.3 (4.5)microg/L and the median was 23.7 microg/L; at 24 months, these values were 21.1 (6.2) microg/L and 20.3 microg/L, respectively. Twelve- and 24-month manganese concentrations were correlated (Spearman correlation = 0.55) and levels declined over time ([beta] = -5.7 [95% CI = -6.2 to -5.1]). We observed an inverted U-shaped association between 12-month blood manganese and concurrent mental development scores (compared with the middle 3 manganese quintiles, for the lowest manganese quintile, [beta] = -3.3 [-6.0 to -0.7] and for the highest manganese quintile, [beta] = -2.8 [-5.5 to -0.2]). This 12-month manganese effect was apparent but diminished with mental development scores at later ages. The 24-month manganese levels were not associated with neurodevelopment. CONCLUSIONS: These results suggest a possible biphasic dose-response relationship between early-life manganese exposure at lower exposure levels and infant neurodevelopment. The data are consistent with manganese as both an essential nutrient and a toxicant.

Kern, C. H., G. D. Stanwood, et al. (2010). **Preweaning manganese exposure causes hyperactivity, disinhibition, and spatial learning and memory deficits associated with altered dopamine receptor and transporter levels.** Synapse **64**(5): 363-378. [HYPERLINK "http://www.ncbi.nlm.nih.gov/pubmed/20029834"]

Abstract: Epidemiological studies in children have reported associations between elevated dietary manganese (Mn) exposure and neurobehavioral and neurocognitive deficits. To better understand the relationship between early Mn exposure and neurobehavioral deficits, we treated neonate rats with oral Mn doses of 0, 25, or 50 mg Mn/kg/day over postnatal day (PND) 1–21, and evaluated behavioral performance using open arena (PND 23), elevated plus maze (PND 23), and 8-arm radial maze (PND 33–46) paradigms. Brain dopamine D1 and D2-like receptors, and dopamine transporter (DAT) densities were determined on PND 24, and blood and brain Mn levels were measured to coincide with behavioral testing (PND 24, PND 36). Preweaning Mn exposure caused hyperactivity and behavioral disinhibition in the open arena, but no altered behavior in the elevated plus maze. Manganese-exposed males committed significantly more reference and marginally more working errors in the radial arm maze compared to controls. Fewer Mn exposed males achieved the radial maze learning criterion, and they required more session days to reach it compared to controls. Manganese-exposed animals also exhibited a greater frequency of stereotypic response strategy in searching for the baited arms in the maze. These behavioral and learning deficits were associated with altered expression of the dopamine D1 and D2 receptors and the DAT in prefrontal cortex, nucleus accumbens, and dorsal striatum. These data corroborate epidemiological studies in children, and suggest that exposure to Mn

during neurodevelopment significantly alters dopaminergic synaptic environments in brain nuclei that mediate control of executive function behaviors, such as reactivity and cognitive flexibility.

Background: Recent evidence suggests that low-level environmental exposure to manganese adversely affects child growth and neurodevelopment. Previous studies have addressed the effects of prenatal exposure, but little is known about developmental effects of early postnatal exposure.

Methods: We studied 448 children born in Mexico City from 1997 through 2000, using a longitudinal study to investigate neurotoxic effects of early-life manganese exposure. Archived blood samples, collected from children at 12 and 24 months of age, were analyzed for manganese levels using inductively coupled plasma mass spectrometry. Mental and psychomotor development were scored using Bayley Scales of Infant Development at 6-month intervals between 12 and 36 months of age.

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Conclusions: These results suggest a possible biphasic dose-response relationship between early-life manganese exposure at lower exposure levels and infant neurodevelopment. The data are consistent with manganese as both an essential nutrient and a toxicant. (*Epidemiology* 2010;21: 433–439)

Background: Excessive exposure to manganese (Mn), an essential trace element, has been shown to be neurotoxic, especially when inhaled. Few studies have examined potential effects of Mn on cognitive functions of environmentally exposed children.

Objective: This study was intended to estimate environmental exposure to Mn resulting from mining and processing and to explore its association with intellectual function of school-age children.

Methods: Children between 7 and 11 years of age from the Molango mining district in central Mexico (n = 79) and communities with similar socioeconomic conditions that were outside the mining district (n = 93) participated in the cross-sectional evaluation. The revised version of the

Wechsler Intelligence Scale for Children adapted for the Mexican population was applied. Concentrations of Mn in blood (MnB) and hair (MnH) were used as biomarkers of exposure. Results: Exposed children had significantly higher median values for MnH (12.6 µg/g) and MnB (9.5 µg/L) than did nonexposed children (0.6 µg/g and 8.0 µg/L, respectively). MnH was inversely associated with Verbal IQ [$\beta = -0.29$; 95% confidence interval (CI), -0.51 to -0.08], Performance IQ ($\beta = -0.08$; 95% CI, -0.32 to 0.16), and Total Scale IQ ($\beta = -0.20$; 95% CI, -0.42 to 0.02). MnB was inversely but nonsignificantly associated with Total and Verbal IQ score. Age and sex significantly modified associations of MnH, with the strongest inverse associations in young girls and little evidence of associations in boys at any age. Associations with MnB did not appear to be modified by sex but appeared to be limited to younger study participants. Conclusions: The findings from this study suggest that airborne Mn environmental exposure is inversely associated with intellectual function in young school-age children.

C. Kern et al. (2010) **Preweaning Manganese Exposure Causes Hyperactivity, Disinhibition, and Spatial Learning and Memory Deficits Associated With Altered Dopamine Receptor and Transporter Levels** DOI 10.1002/syn.20736 Published online in Wiley InterScience (www.interscience.wiley.com).

ABSTRACT Epidemiological studies in children have reported associations between elevated dietary manganese (Mn) exposure and neurobehavioral and neurocognitive deficits. To better understand the relationship between early Mn exposure and neurobehavioral deficits, we treated neonate rats with oral Mn doses of 0, 25, or 50 mg Mn/kg/day over postnatal day (PND) 1–21, and evaluated behavioral performance using open arena (PND 23), elevated plus maze (PND 23), and 8-arm radial maze (PND 33–46) paradigms. Brain dopamine D1 and D2-like receptors, and dopamine transporter (DAT) densities were determined on PND 24, and blood and brain Mn levels were measured to coincide with behavioral testing (PND 24, PND 36). Preweaning Mn exposure caused hyperactivity and behavioral disinhibition in the open arena, but no altered behavior in the elevated plus maze. Manganese-exposed males committed significantly more reference and marginally more working errors in the radial arm maze compared to controls. Fewer Mn exposed males achieved the radial maze learning criterion, and they required more session days to reach it compared to controls. Manganese-exposed animals also exhibited a greater frequency of stereotypic response strategy in searching for the baited arms in the maze. These behavioral and learning deficits were associated with altered expression of the dopamine D1 and D2 receptors and the DAT in prefrontal cortex, nucleus accumbens, and dorsal striatum. These data corroborate epidemiological studies in children, and suggest that exposure to Mn during neurodevelopment significantly alters dopaminergic synaptic environments in brain nuclei that mediate control of executive function behaviors, such as reactivity and cognitive flexibility.

Menezes-Filho, J. A., M. Bouchard, et al. (2009) **Manganese exposure and the neuropsychological effect on children and adolescents: a review.** Rev Panam Salud Publica 26(6): 541-548. http://www.ncbi.nlm.nih.gov/pubmed/20107709

Abstract: Objectives. Manganese (Mn) is an essential element, but overexposure can have neurotoxic effects.

Methods. In this article, we review and summarize studies on exposure to Mn and nervous system impairments in children.

Results. We identified 12 original articles published between 1977 and 2007. Overexposure to Mn was suspected to occur through diverse sources: infant milk formula, drinking water, industrial pollution, and mining wastes. The most common bioindicator of exposure to Mn was hair Mn content, but some studies measured Mn in blood, urine, or dentin; one study on prenatal exposure measured Mn content in cord blood. Most studies indicate that higher postnatal exposure to Mn is associated with poorer cognitive functions and hyperactive behavior.

Conclusions. The limitations of the existing studies are numerous: most were cross-sectional, had a modest sample size, and lacked adjustment for important confounders. Future investigations should be performed on a larger sample size and include a more detailed exposure assessment, addressing multiple sources of exposure such as food, water, and airborne particulates.

Brown, M. T. and B. Foos (2009). **Assessing Children's Exposures and Risks to Drinking Water Contaminants: A Manganese Case Study.** Human and Ecological Risk Assessment: An International Journal **15**(5): 923-947. http://www.tandfonline.com/doi/full/10.1080/10807030903153030.

Abstract: Compared to adults, children maybe more highly exposed to toxic substances in drinking water because they consume more water per unit of body weight. The U.S. Environmental Protection Agency (USEPA) has developed new guidance for selecting age groups and age-specific exposure factors for assessing children's exposures and risks to environmental contaminants.

Research Aim: To demonstrate the application and importance of applying age-specific drinking water intake rates, health reference values, and exposure scenarios when assessing drinking water exposures because these approaches illustrate the potential for greater potential for adverse health effects among children.

Methods: manganese, an essential nutrient and neurotoxicant, was selected as a case study and chemical of potential concern for children's health. A screening-level risk assessment was performed using age-specific drinking water intake rates and manganese concentrations from U.S. public drinking water systems.

Results: When age-specific drinking water intake rates are used to calculate dose, formula-fed infants receive the highest dose of manganese from drinking water compared to all other age groups. Estimated hazard quotients suggest adverse health effects are possible. Use of USEPA's standardized childhood age groups and childhood exposure factors significantly improves the understanding of childhood exposure and risks.

Ljung, K. S., M. J. Kippler, et al. (2009). **"Maternal and early life exposure to manganese in rural Bangladesh."** Environ Sci Technol **43**(7): 2595-2601. [HYPERLINK "http://pubs.acs.org/doi/abs/10.1021/es803143z"].

Abstract: Manganese exposure and biomarker concentrations during early pregnancy and lactation were investigated in 408 women living in an area with elevated concentrations of both arsenic and manganese in drinking water derived from wells. About 40% of the water samples had manganese concentrations above the World Health Organization's guideline value and

showed a strong inverse correlation with arsenic concentrations. Water manganese was found to correlate to urine concentrations, but not to blood or breast milk concentrations. No correlations were found among manganese concentrations in urine, blood, or breast milk. Compared to other populations, manganese concentrations in both urine and blood, but not breast milk, were elevated in the Bangladeshi women and more similar to those of occupationally exposed groups. The lack of associations with water manganese is likely due to variable exposure via water and food, and differences in bioavailability, as well as a complex and/or strict regulation of intestinal manganese absorption, in turn being influenced by nutritional as well as physiological and genetic factors. The results indicate that elevated maternal manganese exposure does not necessarily lead to exposure of breast-fed infants, stressing the importance of breast feeding in high manganese areas. However, the implications of fetal exposurefrom elevated maternal exposure need further investigation.

Moreno, J. A., E. C. Yeomans, et al. (2009). **"Age-dependent susceptibility to manganese-induced neurological dysfunction."** <u>Toxicol Sci</u> **112**(2): 394-404. doi: 10.1093/toxsci/kfp220.

Abstract: Chronic exposure to manganese (Mn) produces a spectrum of cognitive and behavioral deficits associated with a neurodegenerative disorder resembling Parkinson's disease. The effects of high-dose exposure to Mn in occupational cohorts and in adult rodent models of the disease are well described but much less is known about the behavioral and neurochemical effects of Mn in the developing brain. We therefore exposed C57Bl/6 mice to Mn by intragastric gavage as juveniles, adults, or both, postulating that mice exposed as juveniles and then again as adults would exhibit greater neurological and neurochemical dysfunction than mice not preexposed as juveniles. Age- and sex-dependent vulnerability to changes in locomotor function was detected, with juvenile male mice displaying the greatest sensitivity, characterized by a selective increase in novelty-seeking and hyperactive behaviors. Adult male mice preexposed as juveniles had a decrease in total movement and novelty-seeking behavior, and no behavioral changes were detected in female mice. Striatal dopamine levels were increased in juvenile mice but were decreased in adult preexposed as juveniles. Levels of Mn, Fe, and Cu were determined by inductively coupled plasma-mass spectrometry, with the greatest accumulation of Mn detected in juvenile mice in the striatum, substantia nigra (SN), and cortex. Only modest changes in Fe and Cu were detected in Mn-treated mice, primarily in the SN. These results reveal that developing mice are more sensitive to Mn than adult animals and that Mn exposure during development enhances behavioral and neurochemical dysfunction relative to adult animals without juvenile exposure.

Menezes-Filio et al (2009) **Elevated manganese and cognitive performance in school-aged children and their mothers** Environ Res. 2011 January ; 111(1): 156–163. doi:10.1016/j.envres.2010.09.006.

Background—Growing evidence suggests that excess manganese (Mn) in children is associated with neurobehavioral impairments. In Brazil, elevated hair Mn concentrations were reported in children living near a ferro-manganese alloy plant.
Objectives—We investigated these children's and caregivers' cognitive function in relation to bioindicators of Mn exposure.
Methods—In this cross-sectional study, the WISC-III was administered to 83 children aged

between 6 and 12 years; the Raven Progressive Matrix was administered to the primary caregivers
(94% mothers), who likewise responded to a questionnaire on socio demographics and birth history. Mn in hair (MnH) and blood (MnB) and blood lead (PbB) were measured by graphite furnace atomic absorption spectrometry (GFAAS).
Results—Children's mean MnB and MnH were 8.2 μg/L (2.7 – 23.4) and 5.83 μg/g (0.1 −86.68), respectively. Mean maternal MnH was 3.50 μg/g (0.10 – 77.45) and correlated to children's MnH (rho=0.294, p=0.010). Children's MnH was negatively related to Full-Scale Intelligence Quotient (IQ) and Verbal IQ; β coefficients for MnH were −5.78 (95% CI −10.71 to −0.21) and −6.72 (−11.81 to −0.63), adjusted for maternal education and nutritional status. Maternal MnH was negatively associated with performance on the Raven's (β = −2.69, 95% CI −5.43 to 0.05), adjusted for education years, family income and age.
Conclusions—These findings confirm that high MnH in children is associated with poorer cognitive performance, especially in the verbal domain. Primary caregiver's IQ is likewise associated to Mn exposure, suggesting that, in this situation, children's cognition may be affected directly and indirectly by Mn exposure.

"Co-exposure to environmental lead and manganese affects the intelligence of school-aged children." Neurotoxicology **30**(4): 564-571. [HYPERLINK "http://www.ncbi.nlm.nih.gov/pubmed/19635390"].

Abstract: BACKGROUND: Exposure to environmental levels of lead (Pb) and manganese (Mn) has been associated with detrimental effects to neurodevelopment. However, little is known about the potential association between environmental levels of Pb and Mn on intelligence of children. The aims of the study were to investigate the association of community level of Pb and Mn with the intelligence of school-aged children, and to explore the implications of joint exposure to these two heavy metals.
METHODS: A cross-sectional examination of blood Pb and Mn concentrations was performed, and the intelligence quotient (IQ) was determined for 261 Korean children aged 8-11 years.
RESULTS: The mean blood concentrations of Pb and Mn were 1.73 microg/dL (SD=0.8; median=1.55; range=0.42-4.91) and 14.3 microg/L (SD=3.8; median=14.0; range=5.30-29.02), respectively. Both Pb and Mn showed significant linear relationship with full-scale IQ (Pb, beta=-0.174, p=0.005; Mn, beta=-0.123, p=0.042) and verbal IQ (Pb, beta=-0.187, p=0.003; Mn, beta=-0.127, p=0.036). Blood Pb (DeltaR(2)=0.03) and Mn (DeltaR(2)=0.01) explained 4% of the variances of the full-scale IQ and 5% of the variances of the verbal IQ. When Pb and Mn levels were entered as predictive variables, additive increase in the explained variances was observed. Finally, full-scale IQ and verbal IQ of the children with blood Mn>14 microg/L showed significant association with Pb, whereas group with Mn<14 microg/L did not, suggesting effect modification between Pb and Mn.
CONCLUSIONS: The present study suggests the presence of additive interaction and effect modification between Pb and Mn on the intelligence of school-aged children, suggesting more attention should be paid to preventing the exposure of disadvantaged children to various combinations of toxic materials.

Ljung, K. and M. Vahter (2007). **Time to re-evaluate the guideline value for manganese in drinking water?** Environ Health Perspect **115**(11): 1533-1538.
http://www.ncbi.nlm.nih.gov/pubmed/18007980

OBJECTIVE: We reviewed the scientific background for the current health-based World Health Organization (WHO) guideline value for manganese in drinking water.
DATA SOURCES AND EXTRACTION: The initial starting point was the background document for the development of the WHO's guideline value for manganese in drinking water as well as other regulations and recommendations on manganese intake levels. Data referred to in these documents were traced back to the original research papers. In addition, we searched for scientific reports on manganese exposure and health effects. DATA SYNTHESIS: The current health-based guideline value for manganese in drinking water is based partly on debatable assumptions, where information from previous reports has been used without revisiting original scientific articles. Presently, preparation of common infant formulas with water containing manganese concentrations equivalent to the WHO guideline value will result in exceeding the maximum manganese concentration for infant formula. However, there are uncertainties about how this maximum value was derived. Concurrently, there is increasing evidence of negative neurologic effects in children from excessive manganese exposure.
CONCLUSIONS: The increasing number of studies reporting associations between neurologic symptoms and manganese exposure in infants and children, in combination with the questionable scientific background data used in setting the manganese guideline value for drinking water, certainly warrant a re-evaluation of the guideline value. Further research is needed to understand the causal relationship between manganese exposure and children's health, and to enable an improved risk assessment.

Bouchard, M., F. Laforest, et al. (2007). **Hair manganese and hyperactive behaviors: pilot study of school-age children exposed through tap water.** Environ Health Perspect **115**(1): 122-127. [HYPERLINK "http://www.ncbi.nlm.nih.gov/pubmed/17366831"]

Abstract: BACKGROUND: Neurotoxic effects are known to occur with inhalation of manganese particulates, but very few data are available on exposure to Mn in water. We undertook a pilot study in a community in Quebec (Canada) where naturally occurring high Mn levels were present in the public water system. Our objective was to test the hypothesis that greater exposure to Mn via drinking water would be reflected in higher Mn content in hair which, in turn, would be associated with increased level of hyperactive behaviors. METHODS: Forty-six children participated in the study, 24 boys and 22 girls, 6-15 years of age (median, 11 years). Their homes received water from one of two wells (W) with different Mn concentrations: WI: mean 610 microg/L; W2: mean 160 pg/L. The Revised Conners' Rating Scale for parents (CPRS-R) and for teachers (CTRS-R) were administered, providing T-scores on the following subscales: Oppositional, Hyperactivity, Cognitive Problems/Inattention, and ADHD Index. RESULTS: Children whose houses were supplied by WI had higher hair Mn (MnH) than those supplied by W2 (mean 6.2+/-4.7 microg/g vs. 3.3+/-3.0 microg/g, p = 0.025). MnH was significantly associated with T-scores on the CTRS-R Oppositional (p = 0.020) and Hyperactivity (p = 0.002) subscales, after adjustment for age, sex, and income. All children with Oppositional and Hyperactivity T-scores > 65 had MnH > 3.0 microg/g. CONCLUSIONS: The

findings of this pilot study are sufficiently compelling to warrant more extensive investigations into the risks of Mn exposure in drinking water.

Erikson, K. M., K. Thompson, et al. (2007). **Manganese neurotoxicity: a focus on the neonate.** Pharmacol Ther **113**(2): 369-377. http://www.ncbi.nlm.nih.gov/pubmed/17084903

Abstract: Manganese (Mn) is an essential trace metal found in all tissues, and it is required for normal amino acid, lipid, protein, and carbohydrate metabolism. While Mn deficiency is extremely rare in humans, toxicity due to overexposure of Mn is more prevalent. The brain appears to be especially vulnerable. Mn neurotoxicity is most commonly associated with occupational exposure to aerosols or dusts that contain extremely high levels (>1-5 mg Mn/m(3)) of Mn, consumption of contaminated well water, or parenteral nutrition therapy in patients with liver disease or immature hepatic functioning such as the neonate. This review will focus primarily on the neurotoxicity of Mn in the neonate. We will discuss putative transporters of the metal in the neonatal brain and then focus on the implications of high Mn exposure to the neonate focusing on typical exposure modes (e.g., dietary and parenteral). Although Mn exposure via parenteral nutrition is uncommon in adults, in premature infants, it is more prevalent, so this mode of exposure becomes salient in this population. We will briefly review some of the mechanisms of Mn neurotoxicity and conclude with a discussion of ripe areas for research in this underreported area of neurotoxicity.

Wasserman, G. A., X. Liu, et al. (2006). **Water manganese exposure and children's intellectual function in Araihazar, Bangladesh.** Environ Health Perspect **114**(1): 124-129. [HYPERLINK "http://www.ncbi.nlm.nih.gov/pubmed/16393669"]

Abstract: Exposure to manganese via inhalation has long been known to elicit neurotoxicity in adults, but little is known about possible consequences of exposure via drinking water. In this study, we report results of a cross-sectional investigation of intellectual function in 142 10-year-old children in Araihazar, Bangladesh, who had been consuming tube-well water with an average concentration of 793 microg Mn/L and 3 microg arsenic/L. Children and mothers came to our field clinic, where children received a medical examination in which weight, height, and head circumference were measured. Children's intellectual function was assessed on tests drawn from the Wechsler Intelligence Scale for Children, version III, by summing weighted items across domains to create Verbal, Performance, and Full-Scale raw scores. Children provided urine specimens for measuring urinary As and creatinine and were asked to provide blood samples for measuring blood lead, As, Mn, and hemoglobin concentrations. After adjustment for sociodemographic covariates, water Mn was associated with reduced Full-Scale, Performance, and Verbal raw scores, in a dose-response fashion; the low level of As in water had no effect. In the United States, roughly 6% of domestic household wells have Mn concentrations that exceed 300 microg Mn/L, the current U.S. Environmental Protection Agency lifetime health advisory level. We conclude that in both Bangladesh and the United States, some children are at risk for Mn-induced neurotoxicity.

US EPA (2004). **Drinking Water Health Advisory for Manganese.** EPA-822-R-04-003, US EPA, Office of Water, Health and Ecological Criteria Division. http://www.epa.gov/ogwdw/ccl/pdfs/reg_determine1/support_cc1_magnese_dwreport.pdf

Woolf, A., R. Wright, et al. (2002). **A Child with Chronic Manganese Exposure from Drinking Water.** <u>Environ Health Perspect</u> **110**(6). [HYPERLINK "http://www.ncbi.nlm.nih.gov/pubmed/12055054"]

Abstract: The patient's family bought a home in a suburb, but the proximity of the house to wetlands and its distance from the town water main prohibited connecting the house to town water. The family had a well drilled and they drank the well water for 5 years, despite the fact that the water was turbid, had a metallic taste, and left an orange-brown residue on clothes, dishes, and appliances. When the water was tested after 5 years of residential use, the manganese concentration was elevated (1.21 ppm; U.S. Environmental Protection Agency reference, < 0.05 ppm). The family's 10-year-old son had elevated manganese concentrations in whole blood, urine, and hair. The blood manganese level of his brother was normal, but his hair manganese level was elevated. The patient, the 10-year-old, was in the fifth grade and had no history of learning problems; however, teachers had noticed his inattentiveness and lack of focus in the classroom. Our results of cognitive testing were normal, but tests of memory revealed a markedly below-average performance: the patient's general memory index was at the 13th percentile, his verbal memory at the 19th percentile, his visual memory at the 14th percentile, and his learning index at the 19th percentile. The patient's free recall and cued recall tests were all 0.5-1.5 standard deviations (1 SD = 16th percentile) below normal. Psychometric testing scores showed normal IQ but unexpectedly poor verbal and visual memory. These findings are consistent with the known toxic effects of manganese, although a causal relationship cannot necessarily be inferred.

Institute of Medicine (2001). **DIETARY REFERENCE INTAKES FOR Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc.** Washington, DC, National Academy Press. [HYPERLINK "http://books.nap.edu/openbook.php?record_id=10026"]

Additional Manganese References (no copies of articles were obtained).

Manganese mixture inhalation is a reliable Parkinson disease model in rats[HYPERLINK \l "_ENREF_1" \o "Sanchez-Betancourt, 2012 #15"]

Abstract

Manganese (Mn) is an essential trace metal. Regardless of its essentiality, it has been reported that the overexposure causes neurotoxicity manifested as extrapyramidal symptoms similar to those observed in Parkinson disease (PD). Recently, our group reported that mice that inhaled for 5 months the mixture of manganese chloride ($MnCl_2$) and manganese acetate $Mn(OAc)_3$ developed movement abnormalities, significant loss of substantia nigra compacta (SNc) dopaminergic neurons, dopamine depletion and improved behavior with l-DOPA treatment. However, this model has only been characterized in mice. In order to have a well-supported and generalizable model in rodents, we used male Wistar rats that inhaled a mixture of 0.04 M

MnCl2 and 0.02 M Mn(OAc)3, 1 h three times a week for 6 months. Before Mn exposure, animals were trained to perform motor tests (Beam-walking and Single-pellet reaching tasks) and were evaluated each week after the exposure. The mixture of MnCl2/Mn(OAc)3 caused alterations in the motor tests, 75.95% loss of SNc dopaminergic neurons, and no cell alterations in Globus Pallidus or striatum. With these results we conclude that the inhalation of the mixture of Mn compounds is a useful model in rodents for the study of PD. â° Manganese inhalation as a Parkinson disease model is progressive and bilateral which makes it more reliable. â° This model has only been characterized in mice. â° In order to have a well-supported and generalizable model in rodents, we used rats that inhaled a mixture of Manganese compounds. â° The mixture of Manganese compounds in rats caused motor alterations and 75.95% loss of dopaminergic neurons. â° The inhalation of the mixture of Mn compounds is a useful model in rodents for the study of PD.

Abstract

Manganese neurotoxicity is linked primarily to inhalation exposure, and its clinical features are almost totally based on high doses, such as those experienced by miners. Manifestations of lower level exposures can take two forms. One is the appearance of neurobehavioral deficits. A second, equally subtle, form is as a promoter, borrowing the term used in carcinogenesis, of neurodegenerative disease. Such low-level environmental exposures may be more potent than expected if they occur as ultrafine particles able to penetrate directly into the brain. The neurological disorder linked most closely to manganese is Parkinson's disease (PD). Although most observers recognize that the features of manganese-induced parkinsonism differ from those of idiopathic PD, they overlap considerably. The overlaps should be expected because the underlying lesions, although distinguishable, are closely linked because they belong to structures with complex interdependent circuitry. Such interdependence makes it feasible to undertake an analysis of how manganese neurotoxicity might elevate the risks of PD. A relatively small increment in risk, expressed as a leftward shift in the age prevalence of PD, incurs significant economic costs.

[ADDIN EN.REFLIST]

Detection and Evaluation of Elevated Lead Release from Service Lines: A Field Study

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⊗ Supporting Information

ABSTRACT: Comparative stagnation sampling conducted in 32 homes in Chicago, Illinois with lead service lines demonstrated that the existing regulatory sampling protocol under the U.S. Lead and Copper Rule systematically misses the high lead levels and potential human exposure. Lead levels measured with sequential sampling were highest within the lead service lines, with maximum values more than four times higher than Chicago's regulatory compliance results using a first-draw sampling protocol. There was significant variability in lead values from different points within individual lead service lines and among different lead service line sites across the city. Although other factors could also influence lead levels, the highest lead results most often were associated with sites having known disturbances to the lead service lines. This study underscores the importance and interdependence of sample site selection, sampling protocol, and other factors in assessing lead levels in a public water system.
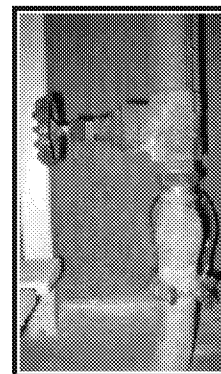
■ INTRODUCTION

Background. Most lead in drinking water comes from premise plumbing materials and lead service lines (LSLs). LSLs are generally the largest source of lead in drinking water when they are present in public water systems.[1] The 1986 Safe Drinking Water Act Amendments banned new lead pipes in the potable water network, but a legacy of millions of partial or whole LSLs remains in many public water systems.[2] Where the term "lead corrosion" is used, it refers to the corrosion of lead plumbing materials that result in the transfer of dissolved or particulate lead into the drinking water.

The Lead and Copper Rule (LCR) sampling is intended to measure the lead levels in drinking water to assess the effectiveness of corrosion control treatment utilized by public water systems (PWSs) to minimize lead in drinking water. PWSs are required to use sampling sites that are presumed to be the highest-risk sites for lead release, and to optimize corrosion control to minimize lead levels at consumers' taps. Most published sampling studies typically focus on systems having high lead levels or systems that have experienced challenges in attempting to balance LCR compliance with various other treatment or water quality objectives. Except for LCR compliance data, little published data exists or is available for systems that are considered to be operating with optimal corrosion control and meeting the lead action level (AL) in the LCR. This study focuses on a system that is considered to have optimized corrosion control using a blended phosphate, with a relatively stable water quality, and compliance results historically well below the lead AL. This situation is representative of a large percentage of systems serving 100,000 or more people that utilize orthophosphate or blended phosphates for corrosion control and the vast majority of

systems are meeting the lead AL based on the current sampling protocol in the LCR. Additional information on the LCR and study is available in the Supporting Information (SI). This study focused on whether (1) the current LCR compliance sampling protocol adequately captures the peak lead levels in a water system; (2) "preflushing" (PF) results in capturing lower lead levels in samples compared to samples collected under normal household usage (NHU) conditions; (3) a first-draw sampling protocol appropriately determines the adequacy of optimal lead corrosion control in water systems with LSLs; and (4) there is seasonal variability in the sampling results using the different sampling protocols.

System Information. The Chicago Department of Water Management (CDWM) operates two similar conventional surface water filtration treatment plants serving approximately 5.4 million residents, including those in 125 suburbs. Lake Michigan is the sole water source, with relatively stable water quality leaving the treatment plants and in the distribution system (Table 1). Before the LCR, CDWM utilized pH/alkalinity adjustment for corrosion control. CDWM switched to a proprietary blended phosphate at both plants between 1993 and 1994 which is still used as the primary corrosion control treatment.

The LCR requires public water systems to collect lead samples using a first-draw (FD) sampling protocol, and samples were collected almost exclusively from single-family homes with LSLs as required by the LCR sample site selection require-

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Table 1. Water Quality Data 2011

parameter	outlets		distribution	
	min	max	min	max
temp (°C)	4	24	5	23
turbidity (NTU)	0.1	0.2	0.1	0.4
pH	7.5	7.8	7.7	7.8
Cl$_2$ residual (mg/L)	1.0	1.2	0.7	0.9
total alkalinity (mg/L as CaCO$_3$)	103	108	98	108
chloride (Cl, mg/L)	16	20	17	20
sulfate (mg/L)	29	31	29	30
Ca (mg/L)	34	39	34	39
PO$_4$ (mg/L)	0.4	0.6	0.5	0.5
total PO$_4$ (mg/L)	0.8	1.1	0.8	1.2
Al (μg/L)	34	126	29	113
Fe (μg/L)	<5	<5	<5	34
Mn (μg/L)	<3	<3	<3	<3

ments.[3] Since the initial LCR monitoring, Chicago has exceeded the lead AL only once, during July—December 1992, with an average 90th percentile compliance monitoring value between 1999 and 2010 of 6 μg/L (SI Table S2).[3]

The LCR requires 1-L, FD tap samples of water that has stood motionless in the plumbing system (i.e., has stagnated within the plumbing) for at least 6 h. The two variants of the FD sampling protocol currently used by public water systems are defined herein as the NHU first-draw sample, where water is used in a normal household manner, and then allowed to sit motionless in the plumbing for at least 6 h before the sample is collected; and the PF first-draw sample, where the water is run from the sampling tap for a specified amount of time immediately prior to the stagnation period. However, the LCR does not provide specific details on water use during the stagnation period.

Almost all PWSs in the U.S. rely on residents to collect compliance samples under the LCR and there are differences across the U.S. in how systems instruct residents not to use the water during the stagnation period prior to collecting the sample. A review of example sets of sampling instructions provided to residents by large PWSs in the U.S. found that some are instructed not to use any water *from the tap to be sampled* during the stagnation period. Others are instructed not to use *any water in the household.* Prior to 2009, CDWM used the PF first-draw sampling protocol, with a 5-min preflush preceding stagnation. Recent instructions to residents included not using water from the sampling tap or from any nearby tap until the (poststagnation) samples were collected, and to collect samples as soon as possible after the minimum required 6-h stagnation period. Regardless of the sampling protocol, resident-collected samples necessitate the use of simple instructions and make it difficult to ensure strict adherence to any sampling protocol. In addition, the diverse premise plumbing materials and configurations (SI Table S1) represent varying effects of flow rates, hydraulic flow characteristics, and possible lead sorption/particle release effects on the shapes of the lead profiles, particularly with corroded galvanized pipe locations.[4,5]

■ MATERIALS AND METHODS

Sampling Objectives and Protocol. Since the promulgation of the LCR, new research on lead corrosion has shown that there are many mechanisms and water quality factors

involved.[1,4,6−11] Specifically, the sampling protocols used in this study were evaluated to determine if

- preflushing biases results;
- first-draw samples, with or without preflushing, capture the "worst-case" level of lead corrosion under normal use conditions; and
- seasonal variability affects lead concentrations (in this water system).

Consistent with the LCR requirements and CDWM compliance sampling, samples for this study were collected by volunteer residents from 32 single-family residences, built between 1890 and 1960, with LSLs. An additional 5 homes were sampled and determined not to have LSLs, and were therefore excluded from further sampling. All results are included in the Supporting Information, but the non-LSL sites were not used in the data analysis (SI Tables S4a, S5, S6a, S6b, and S7).

Information was requested on the specific plumbing configurations of each sampling site to a much greater extent than the regulatory requirements which simply require the plumbing material to be identified. This information, along with analyses conducted for lead, copper, iron, and zinc for each sample, facilitated a better understanding of the observed water lead levels. Residents were asked to (1) complete a plumbing profile identifying the kitchen tap and meter or internal shut-off valve, and (2) describe the internal plumbing, including any recent plumbing work (SI Figure S1). The information provided by residents along with the results of the four metals provided additional information on the sequences of plumbing materials, and the presence of in-line brass plumbing components. CDWM provided the locations of water mains, service line materials, work conducted by the city at each residence (meter installation or repair, shut-off valve repair/ replacement, service line leak repair, street excavation), and monthly water use data for residences with water meters. The information provided by CDWM on water main locations was used to measure the distance from the water main to each residence, and internal plumbing information provided by residents was used along with the measured length from the water main to the residence to approximate the LSL length (SI Table S1).

Residents were provided with written sampling and reporting instructions for each sampling event (SI Figures S41−S45). One-liter, high-density polyethylene (HDPE), wide-mouth (5.5 cm, 2.2 in.) sample bottles were used to collect all samples. Residents were instructed not to remove aerators prior to sampling and not to collect samples after point-of-use or point-of-entry treatment devices.

Several prior studies have suggested that significant contributions of particulate-associated lead can be mobilized as a function of flow rate and turbulence in certain water chemistries, though studies have not developed predictive relationships to premise plumbing material, scale composition, and hydraulic flow characteristics.[6,10−15] To try to achieve the most aggressive high flow conditions under realistic field conditions, residents were instructed to collect all samples by slowly opening the cold water kitchen tap until fully open. Upon receipt, the samples were inspected by EPA for visible particulate matter prior to delivery to the laboratory.

For all first-draw samples, residents were instructed not to use any water throughout the household (i.e., no showering, washing clothes/dishes, flushing toilets, etc.) during the

First Draw and 45-Second Flushed Samples
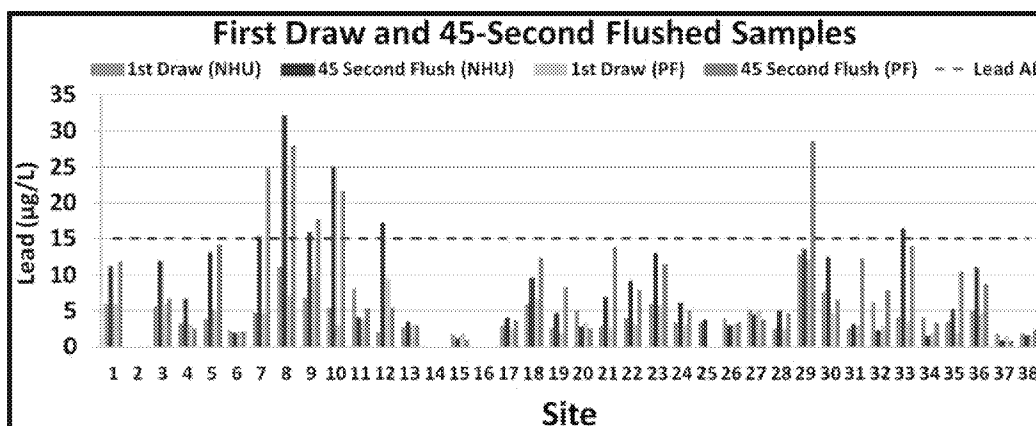
Figure 1. First round lead results for all sites.

minimum mandatory 6-h stagnation period. In this study, PF samples include a flush of at least 5 min prior to the mandatory minimum 6-h stagnation period. A NHU sample had no preflushing prior to the mandatory minimum stagnation period. Residents were instructed to allow the water to sit motionless in the household plumbing a minimum of 6 h, but not more than 24 h, and to record the dates/times the taps were flushed prior to the stagnation period, and the dates/times samples were collected following the stagnation period. First-draw samples using both variants (NHU and PF) were collected in the first and third rounds of monitoring in March/April and September/October, respectively. Additionally, 45-s flushed samples were collected in the first round to evaluate whether a second-draw sample more accurately captured the level of corrosion. Three-min, 5-min, and 7-min flushed samples were collected in the third round of sampling to provide guidance to volunteers when high lead levels were found (SI Table S7). This information can also be used to provide site-specific guidance on minimum flushing times necessary to reduce consumer exposure to lead in drinking water.

In the first round of sampling, each resident collected a NHU first-draw sample and then a second-draw (45-s flushed) sample after allowing the water to run for 45 s. On the second day, residents collected a PF first-draw sample and then a second 45-s flushed sample. EPA's current Public Notification Handbook advises[16] residents to run the water 30 s or until it turns cold before consuming, if the water has not been used for an unspecified "extended period of time", which can result in higher lead levels at the tap for consumers. It has also been previously demonstrated that in some situations, this advice can cause residents to consume the worst-case water sitting stagnant in the LSL.[17] (Figure 1)

Sites 14, 15, 16, and 37 were verified as not having LSLs and were excluded from further sampling. Site 2 was verified as not having a LSL following the June sequential sampling and was excluded from the final round of monitoring. The 45-s flushed sampling was discontinued following the March/April sampling first round due to the presence of severely corroded galvanized pipe in some of the residences (SI Figure S4) which reduced the inner pipe diameter, restricting water flow and resulting in varying volumes of water flowing through the plumbing for the same flush time.

In June 2011, each resident collected a total of twelve PF sequential samples in one day of sampling. The first PF sequential sample was also the PF first-draw sample for the data analysis. All samples were analyzed for lead, copper, zinc, and

iron. The co-occurrence of the metals, along with plumbing details, was used in qualitative assessments to correlate lead results with potential sources of lead in the plumbing network (SI Figure S6).[4,10]

In September/October 2011, each resident collected a NHU first-draw sample, and a minimum of 11 PF sequential 1-L samples. Sites with high lead levels in the previous rounds collected an additional 3 or 4 PF sequential samples, and one site with a very long LSL (159 ft, 48 m) collected an additional 9 PF sequential samples. The additional PF sequential samples were collected to determine the point at which lead levels consistently dropped below the AL. All samples collected are included in the sampling summary with the numbers and types of samples collected at each site (SI Table S3).

Most stagnation times were relatively consistent across most sites at between 6 and 8.5 h, and all but two sites had stagnation times between 6 and 9 h 10 min, which facilitated unadjusted comparisons (SI Table S6c).

Additional flushed samples were collected in September/October for high lead sites in order to provide residents with guidance on minimizing lead levels in their drinking water. Recommended minimum flushing times were then estimated based on the lead levels and LSL lengths. These results are included in the Supporting Information, but not discussed here.

Sample Analyses. All samples were visually inspected for particulate matter prior to delivery to the EPA Chicago Regional Laboratory. Samples were preserved upon receipt by the laboratory using concentrated nitric acid to pH <2 and held for a minimum of 24 h prior to analysis.[18] The laboratory's Reporting Limits (RL) for lead, copper, and zinc in drinking water samples, using EPA Method 200.8, are 0.5, 1, and 10 $\mu g/L$, respectively. The laboratory's RL for iron in drinking water samples, using EPA Method 200.7, is 80 $\mu g/L$. Additional laboratory information is included in the Supporting Information.

RESULTS AND DISCUSSION

Both Variants of the First-Draw Protocol Significantly Underestimated Peak Lead Levels, and the NHU First-Draw Protocol Yielded Higher Results Overall than the PF First-Draw Protocol. The 90th percentile lead values for all three rounds of first-draw sampling using both variants were slightly higher than Chicago's historical compliance results, but still fell well below the lead AL (SI Table S4b). Only 2% of the total number of first-draw samples (3 of 151) exceeded the AL despite the presence of lead levels well above the lead action
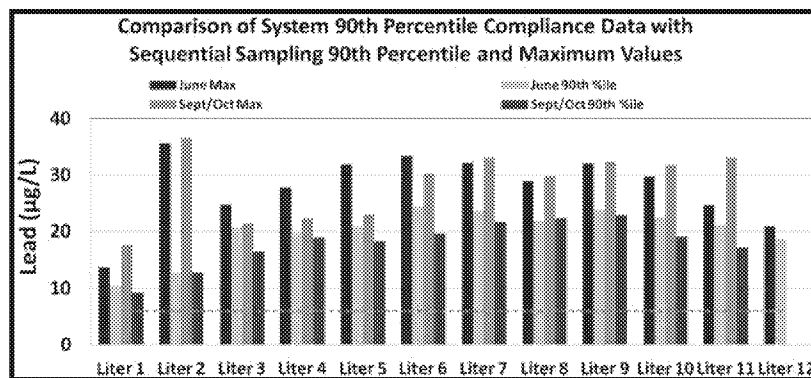
Figure 2. Comparison of 90th percentile LCR compliance data to 90th percentile values from LSL samples (across sites by liter) and maximum values from LSLs. The green dashed line indicates the average 90th percentile compliance monitoring value for Chicago between 1999 and 2010 of 6 μg/L.
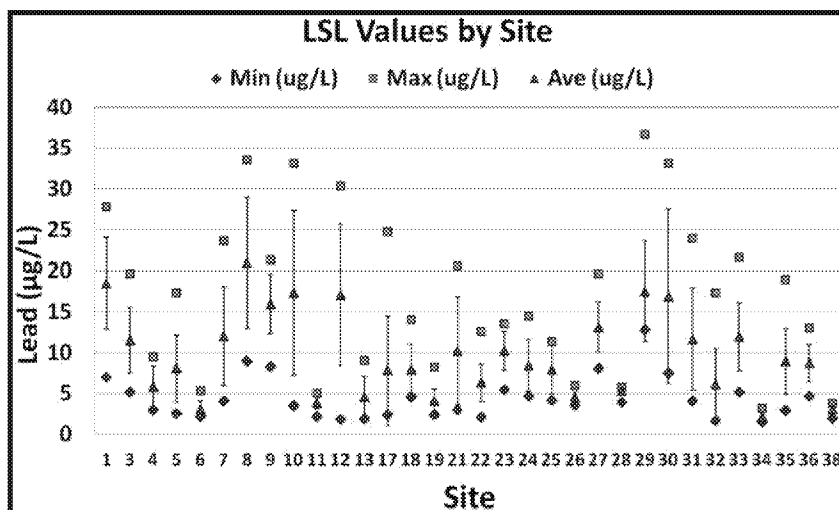
Figure 3. LSL results were highly variable within each LSL and from site to site. Error bars represent 1 standard deviation.

level within the service lines as indicated by the 45-s flushed results in the first round of monitoring and sequential sampling results in the second and third rounds.

In contrast, if the 90th percentile value of each of the successive sequential liter samples from the LSLs is computed across all sampling sites, the lead levels were up to four times higher than Chicago's average 90th percentile value using FD samples. Some peak values for each sequential liter calculated across all sampling sites were over twice the lead AL and up to six times higher than the regulatory compliance data (Figure 2). In summary, 69 of 336 (21%) of the individual sequential samples collected in June and 75 of 319 (24%) of sequential samples in September/October exceeded the lead AL, indicating that current sampling protocols will often considerably underestimate the peak lead levels and overall mobilized mass of waterborne lead in a system with lead service lines.

The NHU results were numerically higher overall than the corresponding PF values for most sites, but the differences were not statistically significant. The PF first-draw protocol produced lower individual results than NHU first-draw protocol in 23 of 32 sample pairs in March/April, and 20 of 27 sample pairs in Sept/Oct (SI Table S4a). Although NHU first-draw samples were collected without directing the residents to flush the tap prior to the stagnation period, NHU can involve showering, washing dishes, or doing laundry a short time prior to the stagnation period, which could clear the lead from the pipes

similar to preflushing the tap. Thus a NHU sample can be effectively the same as a PF sample and yield similar results. Since the sequential sampling results from these same sites show that there is much higher lead present within the LSL at the same time that the NHU and PF first-draw samples were collected, it stands to reason that if the NHU activities were not undertaken, and a larger sample set were used, the NHU samples would yield results that were statistically higher than the corresponding PF samples. The distance from the kitchen tap to the beginning of the LSL was highly variable, ranging from approximately 3 to 87 feet (0.9 to 27 m), and the measured LSL lengths ranged from 43 to 159 feet (13 to 48 m). Consequently, for sites with shorter total plumbing lengths, the initial and final sequential samples would include relatively uncontaminated water from the water main following the 5-min tap preflushing. These samples would contain little to no LSL lead contribution, consistent with plumbosolvency and radial diffusion/flow principles.[5,19,20] A targeted LSL sampling protocol isolating only LSL contact water would likely yield a higher percentage of results above the lead AL for systems with Pb(II) pipe scale chemistry, but the specific location of the peak lead levels will necessarily vary with premise plumbing configurations.

Seasonal Variability. In a site-by-site comparison, lead concentrations were higher in Sept/Oct than in Mar/Apr or June, with the starkest statistical difference between first-draw
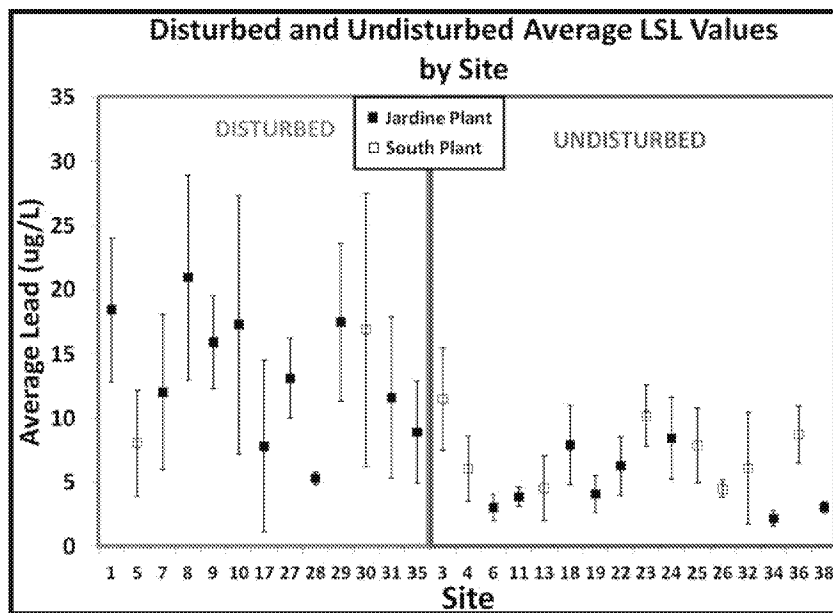
Figure 4. Average lead levels at disturbed and undisturbed sites. Error bars represent 1 standard deviation.

NHU samples collected in Mar/April and Sept/Oct ($p = 0.03$ for two-tailed paired Student's t-test). Overall, 68% and 69% of NHU and PF first-draw samples, respectively, were higher in Sept/Oct than in Mar/Apr, while 55% of paired sequential samples were higher in Sept/Oct than in June. Seasonal variation in lead levels consists of multiple contributing factors from the source water through the premise plumbing which could not be precisely isolated in this study, but the results in this study are consistent with other findings on seasonal variability (SI Table S6d).[21] Factors include (1) water temperature, (2) water chemistry variation, and (3) fluctuations in water usage for Sept/Oct versus June, which could increase or decrease lead levels.[22,23]

Lead Concentrations Vary Throughout Each Individual LSL and among Different LSLs Across the System. There was a high degree of variability in sequential sample results at most sites, some of which could include a particulate-bound component as reflected in spikes in some sequential sampling results (SI Figures S9–S40). For most sites, no individual sample result from within the LSL can *characterize* the lead concentrations at the site. Within the complete sampling profile results, lead levels at most sites ranged from well below to well above the AL (Figure 3). Under the LCR, this would mean that a system would meet the action level and have no additional regulatory requirements or would exceed the AL and be required to implement additional requirements, depending on which sample result is selected as the compliance sample. The variability within sites and between sites is similar in trend to that found in several other studies reporting sequential sampling conducted in water systems with different corrosion control strategies and chemistries from CDWM.[1,4,10,12,14,15,24–27]

Additional compliance data from a second large utility (City B) which exceeded the lead AL and conducted sampling using the temperature change LSL sampling protocol in the LCR,[3] yielded similar variability across the system (SI Figure S8 and Table S9). A total of 1975 LSL sites were sampled, with 1762 results (89%) below the lead AL; 128 results (6.5%) from 16 to 30 μg/L; 57 results (2.8%) from 31 to 50 μg/L; and 28 results (1.4%) between 51 and 580 μg/L. This LSL sampling protocol

is similarly vulnerable to low biases, although many results were considerably higher than the AL (SI Figure S8).

Factors Affecting Lead Levels. The majority of high lead results occurred at sites with a documented physical disturbance of the LSL between 2005 and 2011 (Figure 4). The actual extent to which the LSL was physically disturbed is unknown for all sites, and the records of disturbances are based on information provided by CDWM and by the sampling volunteers (SI Figures S9–S40).

For the purpose of this study a physical LSL disturbance is defined as a meter installation or replacement, autometer-reader (AMR) installation, service line leak repair, external service shut-off valve repair or replacement, or significant street excavation directly in front of the home that could disturb the LSL. An "undisturbed" site is an unmetered site where neither the CDWM nor resident have a record or recollection of any disturbance, as defined above. A third category, "indeterminate", is used for three sites where CDWM has no record of any LSL disturbance, and the resident did not provide a response as to whether there has been any LSL disturbance. Cross-checking was important because information provided by volunteers in some cases contradicted CDWM records, and upon further investigation, the records were found to be incomplete and were corrected, which resulted in reclassification of the site.

Of the 13 disturbed sites, 11 sites had 3 or more sequential sampling results above the lead AL, two sites had 2 results each above the AL, and one site had no results above the AL. Of the 16 sites with no known disturbance, only three sites had any results above the lead AL. In the remaining 3 "indeterminate" sites, 30 of 81 sample results (37%) were above EPA's lead AL (Table 2).

A recent AWWA publication on the state of water infrastructure highlights the need for major infrastructure work.[28] This necessary infrastructure work will potentially increase the incidence of damage to the protective scales within LSLs as this work is performed. Inevitably, these physical LSL disturbances will continue to occur with increased frequency as part of daily routine water system maintenance and nonwater related community infrastructure work.

Table 2. Lead Results for Disturbed, Undisturbed, and Indeterminate Sites[a]

disturbed sites			undisturbed sites			indeterminate sites		
no. sites	no. samples	no. above AL	no. sites	no. samples	no. above AL	no. sites	no. samples	no. above AL
13	327	117	16	372	6	3	81	30
% samples over AL: 36%			% samples over AL: 2%			% samples over AL: 37%		

[a]Most lead results above the AL were found at sites with LSL disturbances. Additional results above the AL were also found at sites where the status of the LSL (disturbed or undisturbed) could not be confirmed. Sites without LSL disturbances had few if any results above the AL.

Possible Implications of Water Conservation and Use.

Information provided by CDWM and volunteers anecdotally suggests that low water usage may also play a role in high lead levels at some sites. Of the four locations with the highest average lead levels, three (Sites 1, 29, and 10) had documented low water usage. Site 1 had average monthly water usage of 3444 gallons (13 037 L) which does not appear to be low usage. However, information provided by the resident indicates that the majority of the monthly water usage occurs during a relatively small number of days during the month when there is a high volume of water usage. Site 29 had average monthly usage of 1826 gallons (6912 L), and Site 10 had an average usage of 1438 gallons/month (5443 L/month). For comparison, the mean single-family household water usage is approximately 8582 gallons/month (32 486 L/month), with a sizable standard deviation.[29]

In two locations (Sites 17 and 5), lead levels decreased with an increase in water usage. As water usage approximately doubled at Sites 17 and 5, maximum lead levels from sequential sampling decreased from 25 to 5.5 μg/L and from 17 to 12 μg/L, respectively. Although this represents a small set of samples, these observations support the idea that higher lead levels can be associated with low water usage.[30]

Extrapolating from prior research suggests the necessity of consistent flow to deliver corrosion inhibitor effectively into passivating films,[31] and correlates increased inhibitor dosages with reduced lead release.[10,32−35] Low water usage may inhibit healing of the damaged scales, and influence the rate of galvanic corrosion. Water usage effects cannot be separated from other *seasonal* effects in this study, but prior literature and the combined sequential graphs showing entire profiles shifted up or down from the June to Sept/Oct sampling suggest further investigation is warranted (SI Figures S9−S40). As conservation efforts increase, it will become increasingly important to conduct further research on the relationship between water usage and increases in lead levels.

The results in this study also indicate that more appropriate flushing guidance must be developed, based on neighborhood and premise plumbing characteristics, and whether a home has a LSL or not. Much of the current published and web-based flushing guidance inadvertently increases the risk of exposure to elevated lead levels by clearing an insufficient amount of water volume.[17] Even fully flushing LSLs may only lower lead levels to a limiting, measurable lead level, that relates to the plumbosolvency of the water, the flow rate, the length and internal diameter of the pipe,[5−7,10,19,20] and possibly effects of prior disturbances (SI Table S7).

Risk Identification and Management.

Recently, CDC issued a health alert associating higher elevated blood lead levels with partial LSL replacement,[36] and also concluded that LSLs were an independent risk factor for elevated blood lead levels even when lead levels in drinking water met the LCR lead AL of 0.015 mg/L.[37] As highlighted in this study, LSLs can contribute high lead when they are disturbed in many different ways, not just due to partial LSL replacement, and water usage may also play a role in the resultant high lead levels and potential increased human exposure. In an August 2012 update on lead in drinking water and blood lead levels, the CDC notes that "*The recent recommendations from the CDC Advisory Committee on Childhood Lead Poisoning Prevention to reduce or eliminate lead sources for children before they are exposed underscore the need to reduce lead concentrations in drinking water as much as possible*".[38]

As the ultimate human and environmental health goal, LSLs should be completely removed where possible. The stability of the protective scales within LSLs depends on many factors which can change over time. For example, changes to water quality or treatment have resulted in high lead levels over a sustained period of time (years).[10,39−41] Under the current regulatory framework, elevated lead levels from disturbances, water quality, treatment, or water usage changes can potentially go undetected for up to 3 years between LCR compliance monitoring periods, which can result in increased public exposure over a significant period of time.

Proper selection of sampling sites, sampling protocol, and other site conditions is critical for evaluating the amount of lead corrosion and release that is occurring in the distribution system. Successful optimization of the plumbosolvency treatment depends on an accurate understanding of the corrosion mechanisms, pipe scale mineralogy and structure, and the consequences of LSL disturbances and water conservation efforts. No published studies could be found that systematically investigated the time and inhibitor doses/water quality adjustments necessary to overcome the disturbances and damage to the lead pipe scales that will be routinely occurring throughout cities across the U.S., as long as full or partial lead service lines remain in service.

Analyses of the Chicago LSL scales by EPA (to be reported elsewhere) reveal that the surface coatings on both lead service line and galvanized interior pipes from CDWM are primarily composed of amorphous aluminum, calcium, and phosphorus-rich deposits, and not crystalline lead(II) (or zinc)-orthophosphate phases that are predicted by conventional divalent lead plumbosolvency theory for orthophosphate dosing.[10,33,42] An understanding of the scales is essential to study and implement procedures and strategies for effective and timely repair of the protective scales damaged by LSL disturbances, and to minimize the public's exposure to high lead levels that can result from damaging the scales. Experimental evaluations are critical when scale compositions fall outside the scope of well-understood predictive corrosion control practices.

■ ASSOCIATED CONTENT

Ⓢ Supporting Information

Additional background information, tabular summaries of sampling results, and graphics. This material is available free of charge via the Internet at http://pubs.acs.org.

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Detection and Evaluation of Elevated Lead Release from Service Lines: A Field Study

(Supporting Information for Manuscript ID: es-2013-003636)

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The supporting information provides additional background information, summaries and graphics for the underlying data used in the study.

Summary of Supplemental Figures and Tables			
Figures		**Tables**	
Figure S1	Plumbing Profile Diagram	Table S1	LSL Lengths
Figure S2	Photograph of LSL Bulb	Table S2	Chicago Compliance Data
Figure S3	Photograph of LSL Segment	Table S3	Summary of Samples Collected
Figure S4	Photograph of Corroded Galvanized Pipe	Table S4a	First-draw and Second-draw (45 second flushed) Sample Results
Figure S5	Sample Site Map and Home Age	Table S4b	Comparison of LCR-equivalent 90th percentile results using alternative first-draw protocols.
Figure S6	Graph of Four Metals for Site 9	Table S5	June Sequential Sampling Results
Figure S7	City B Sampling Instructions	Table S6a	Sept/Oct Sequential Sampling Results Used in Analyses
Figure S8	City B LSL Results	Table S6b	Sept/Oct Supplemental Sequential Sampling Results (Not Used in Analyses)
Figure S9-S40	Sequential Sampling Graphs (Lead)	Table S6c	Summary of stagnation times.
Figure S41	Mar/Apr Sampling Instructions	Table S6d	Seasonal variability effects observed
Figure S42	Mar/Apr Sample Collection and Reporting Form	Table S7	Flushed Sampling Results
Figure S43	June Sampling Instructions and Sample Collection and Reporting Form	Table S8	Summary of Disturbed, Undisturbed and Indeterminate Sites
Figure S44	Sept/Oct Sampling Instructions	Table S9	City B Compliance Data
Figure S45	Sept/Oct Sample Collection and Reporting Form		

Background

The Lead and Copper Rule (LCR) is a treatment technique regulation that requires all public water systems to optimize corrosion control and utilizes tap sampling for lead and copper to determine whether additional actions need to be taken by the system. It is important to note that the sampling conducted under the LCR is not designed to evaluate individual consumers' lead exposure or risk and that the lead action level (AL) was not established as a health-based number. The lead AL is the level which EPA determined in 1991 that systems could feasibly meet, taking into account the available treatment technologies and the cost of those treatment technologies. The lead AL should not be viewed or used as a threshold value to determine whether the water is safe or unsafe to drink, and it should be reiterated that the EPA and CDC have determined that there is no safe level of lead exposure (i.e., no level at which there is not an adverse effect).

Tap sampling conducted under the LCR is intended to measure the amount of lead and copper corrosion that is occurring in public water systems using worst-case site selection and a specified sampling protocol. The sampling protocols in the current LCR were established in 1991, based on the existence of many potential sources of lead throughout the water distribution system, including lead service lines connecting the water main to the homes, leaded-solder used to join copper pipe, and leaded-brass devices, such as meters, brass connectors and shut-off valves, faucets and fixtures. The current LCR sampling requirements are prescriptive and based on the relative significance of lead sources in 1991. The sequential sampling protocol (described below, and in the accompanying paper) that resulted in capturing the highest lead, as well as the sample results themselves, are not allowed to be used in the current compliance calculation.

The LCR utilizes a combination of: worst-case site selection (sites expected to yield the highest lead results); sampling protocols used to capture the highest lead; and repeated sampling at the same sites in order to measure the level of lead corrosion that is occurring throughout the water distribution system. Utilizing this sampling structure allows U.S. EPA to keep the sampling burden on public water systems manageable, while still accomplishing the objectives of the sampling under the LCR. Absent these key components, the number of samples needed to accurately assess system-wide corrosion would necessarily need to increase substantially to accomplish the objectives of the LCR.

The action level for lead is 0.015 mg/L, but is presented here as 15 µg/L for the purpose of using consistent units for the data. An exceedance of the lead AL based on the sampling triggers specific actions that a public water system must undertake to protect public health, such as installing or adjusting corrosion control treatment and providing public education. Additionally, where the corrosion control treatment has proven ineffective at lowering lead levels below the lead AL, the removal of lead service lines is triggered. There are many different corrosion mechanisms and factors that govern lead corrosion. The selection of sampling sites, sampling protocol, and site conditions are essential components for evaluating the level of corrosion that is occurring in the distribution system, regardless of the mechanism(s) or contributing factor(s). It is therefore critically important that the sampling protocol accurately portray the level of corrosion that is occurring.

Lead Service Line and Plumbing Information

As part of the sampling protocol, residents were asked to provide a plumbing profile (figure S1), describing their internal plumbing, and identifying the location of the kitchen tap, and shut-off valve/meter.

Volunteer ID: _____

Home Plumbing and Service Line Diagrams

Below there are 4 diagrams for common household plumbing configurations and the 5th diagram is blank. Please review the diagrams and select the diagram that best matches the plumbing configuration for your home. Each of the diagrams shows where the water service line comes into the home and where the kitchen tap is located. If none of the four diagrams matches your home, use the blank diagram (number 5) to draw where the water service line comes into your home and where your kitchen tap is located. If you do not know where the service line comes into the home, you can note that in your Home Plumbing description below.

Note: Some homes have water meters and some do not. On the diagrams below, if you do not have a water meter, pick the diagram that matches where your service line comes into your home and where the kitchen tap is, and cross out the meter symbol ▓

Home Plumbing Description: In the space below, please describe your home plumbing as best you can, from the point at which the water service line comes into your home to the location of your kitchen tap (length of pipe, diameter of pipe, pipe material, etc.):
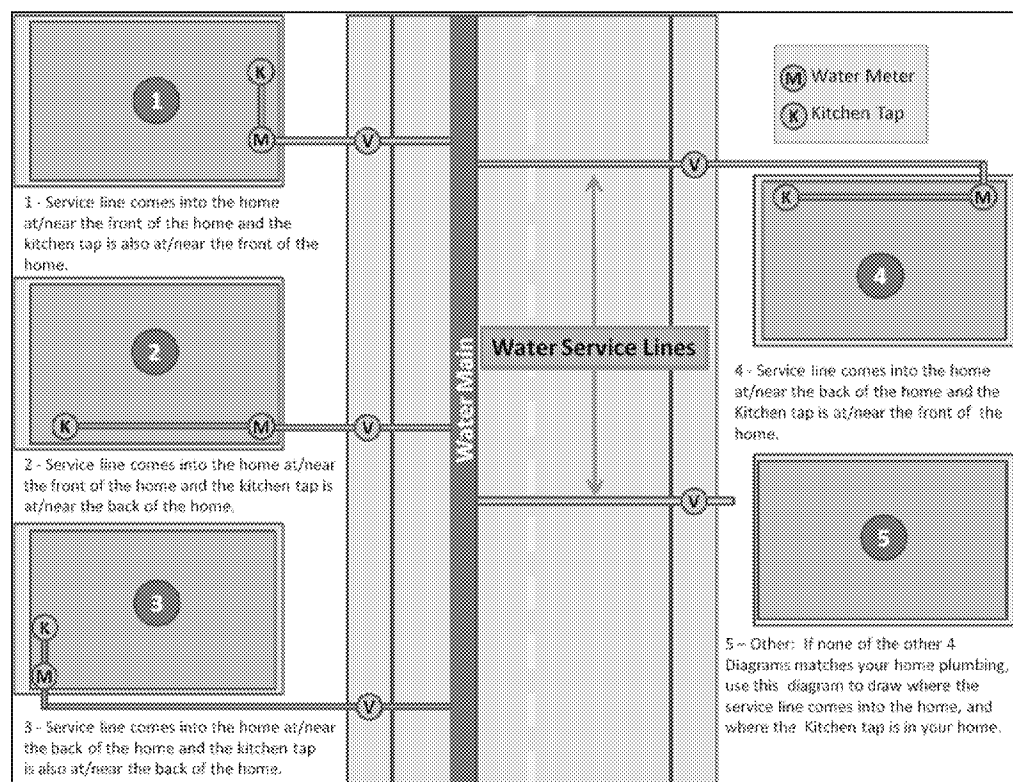
Figure S1: Plumbing Profile Diagram

Table S1 contains a summary of the LSL information for each sampling site. Due to the site-specific plumbing characteristics, the liter which first begins to capture LSL water at each site was expected to be variable, as was the liter which would begin to collect uncontaminated water from the water mains. The study findings regarding whether the current sampling protocol is capturing the corrosion that is occurring are not affected by this limitation.

Site	LSL Length ft (meters)	LSL End Point	Site	LSL Length ft (meters)	LSL End Point
1	89 (27.1)	BFW	22	65 (19.8)	IFW
3	73 (22.3)	IFW	23	66 (20.1)	IFW
4	Unknown	Unknown	24	56 (17.1)	IFW
5	80 (24.4)	IBW	25	70 (21.3)	IFW
6	60 (18.3)	IFW	26	66 (20.1)	IFW
7	59+ (18.0+)	BFW	27	47+ (14.3+)	Unknown
8	57 (17.4)	IFW	28	61+ (18.6+)	Unknown
9	102 (31.1)	BFW	29	159 (48.5)	BFW
10	48+ (14.6+)	IFW	30	49+ (14.9+)	Unknown
11	50 (15.2)	IFW	31	71+ (21.6+)	IFW
12	53 (16.2)	IFW	32	43 (13.1)	IFW
13	49+ (14.9+)	Unknown	33	43+ (13.1+)	IFW
17	58+ (17.7+)	Unknown	34	Unknown	Unknown
18	76 (23.2)	IFW	35	80 (24.4)	BFW
19	63(19.2)	IFW	36	110 (33.5)	IBW
21	46 (14.0)	IFW	38	51 (15.5)	IFW

IFW = LSL ends just inside the front wall
IBW = LSL ends just inside the back wall
BFW = LSL ends at an unknown distance beyond the front wall
+ = Indicates that the LSL was measured from the water main to the front the home, and it is not known whether the LSL extends beyond the front wall of the home.

Table S1: LSL Lengths – The length of the LSLs for most sites were measured and are presented in this table. The LSLs for two sites (site 4 and site 34) were not measured.
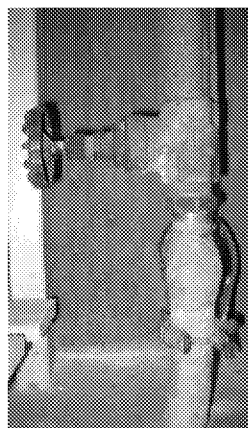
Figure S2: LSL Bulb
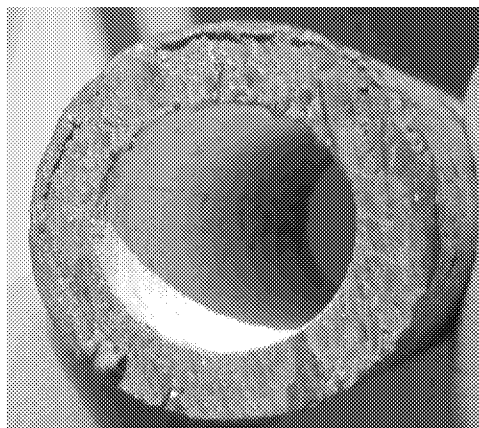
Figure S3: LSL segment (3/4 inch / 1.91 cm diameter)
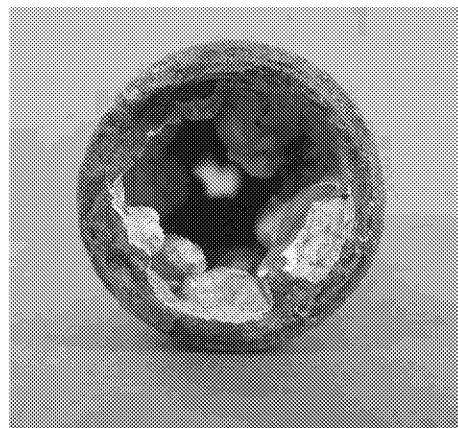
Figure S4: Severely corroded galvanized iron pipe.

Figure S2 shows a typical LSL in Chicago coming up from the foundation of the basement. The lead service line is a dull gray and easily scratched with a key. The soft LSL is typically soldered to the interior (household) plumbing, leaving a characteristic bulb. The LSL can also be connected to household pipe using a brass compression fitting.

Figure S3 is a close-up of a 3/4 inch (1.91 cm) diameter LSL, showing the thickness of a typical LSL.

Figure S4 is a cross-section of a severely corroded galvanized pipe from one of the sample sites. In this photograph the inner diameter is significantly reduced which affects the volume of water that will flow through the pipe in a set amount of time. For homes with corroded galvanized pipe, water will flow slower through the pipe and longer flushing times are generally needed to flush the lead from the plumbing.

City Information

Samples were collected from 32 single-family homes in Chicago with LSLs. Twenty-three homes were in the Jardine Plant service area and nine homes were in the South Plant service area.

Site #	Home Built	Service Area
01	1893	Jardine
03	1960	Jardine
04	1941	South
05	1901	South
06	1953	Jardine
07	1900	Jardine
08	1941	Jardine
09	1920	Jardine
10	1943	Jardine
11	1912	Jardine
12	1952	Jardine
13	1950	South
17	1907	Jardine
18	1953	Jardine
19	1912	Jardine
21	1938	Jardine
22	1924	Jardine
23	1944	South
24	1906	Jardine
25	1917	South
26	1954	South
27	1891	Jardine
28	1932	Jardine
29	1890	Jardine
30	1954	South
31	1923	Jardine
32	1923	South
33	1927	Jardine
34	1915	Jardine
35	1900	Jardine
36	1957	South
38	1927	Jardine

Figure S5: Home age and plant service areas for sampling locations

Table S2 contains a summary of the City's compliance monitoring data for lead. The City exceeded the lead AL only once, during the July-December 1992 compliance monitoring period.

City of Chicago (1992 – 2010) 90th Percentile Lead Values (µg/L)			
Monitoring Period Begin	Monitoring Period End	Number of Samples	90th Percentile Value
1/1/2008	12/31/2010	50	6
1/1/2005	12/31/2007	50	6
1/1/2002	12/31/2004	50	4
1/1/1999	12/31/2001	50	7
1/1/1999	12/31/1999	50	8
1/1/1998	12/31/1998	53	14
7/1/1997	12/31/1997	100	11
1/1/1997	6/30/1997	100	10
1/1/1993	6/30/1993	100	13
7/1/1992	12/31/1992	120	**20**
1/1/1992	6/30/1992	100	10

Table S2: City of Chicago 90th Percentile Compliance Values (1992 – 2010)

Laboratory and Analytical Information

All samples were inspected for visible particulates prior to delivery to the laboratory. In light of the significant increase in visible particulate in the final round of monitoring, the presence of fine particulates that would readily dissolve in the nitric acid preservative should not be discounted. Samples collected during the final round of monitoring coincided with the Fire Department's annual valve exercising. Colloidal lead may explain some of the variability in lead levels between the June and Sept/Oct rounds.

Laboratory blanks, laboratory fortified blanks and laboratory fortified samples were run at a frequency of at least one per twenty samples. Laboratory blanks run with the samples did not have any detections of lead above the reporting limit and all Laboratory fortified blanks and laboratory fortified samples had recoveries greater than 90%.

All laboratory instrumentation was inspected and maintained according to Chicago Regional Laboratory maintenance protocols, and calibrated daily according to Chicago Regional Laboratory standard operating procedures.

The Chicago Regional Lab Quality Assurance (QA) Contact performed a data quality assessment on the results based on laboratory blanks, laboratory fortified blanks and matrix spikes. The QA Contact identified no biases in the sample results due to these quality control measurements.

Sampling Summaries

Sample site summary table - A summary table of the types of samples collected at each site, for each sampling protocol is presented in Table S3 below. The highlighted rows for Sites 2,

14, 15, 16 & 37 were confirmed not to have LSLs and Site 20 is the same residence as Site 21 (Kitchen tap and bathroom tap). Following the first round of sampling, Site 20 (bathroom tap) was no longer sampled, to maintain consistency of using kitchen taps across all sites. Only sample results from LSL sites are presented and analyzed in the study paper. The first liter of the sequential samples in June and Sept/Oct also serve as the PF first-draw samples.

Summary of Samples Collected at Each Site							
Site #	Total # Samples	Mar/April		June	Sept/Oct		
		Day 1	Day 2	Day 1	Day 1	Day 2	Day 3
01	34	A, C	B, D	E-12 samples	A	E-14 samples	F, G, H
02	16	A, C	B, D	E-12 samples	DNS	DNS	DNS
03	30	A, C	B, D	E-12 samples	A	E-11 samples	F, G
04	16	A, C	B, D	E-11 samples	DNS	DNS	DNS
05	28	A, C	B, D	E-12 samples	A	E-11 samples	DNS
06	28	A, C	B, D	E-12 samples	A	E-11 samples	DNS
07	35	A, C	B, D	E-12 samples	A	E-15 samples	F, G, H
08	35	A, C	B, D	E-12 samples	A	E-15 samples	F, G, H
09	30	A, C	B, D	E-12 samples	A	E-11 samples	F, G
10	34	A, C	B, D	E-12 samples	A	E-14 samples	F, G, H
11	30	A, C	B, D	E-12 samples	A	E-11 samples	F, G
12	34	A, C	B, D	E-12 samples	A	E-14 samples	F, G, H
13	16	A, C	B, D	DNS	A	E-11 samples	DNS
14	4	A, C	B, D	DNS	DNS	DNS	DNS
15	4	A, C	B, D	DNS	DNS	DNS	DNS
16	4	A, C	B, D	DNS	DNS	DNS	DNS
17	34	A, C	B, D	E-12 samples	A	E-14 samples	F, G, H
18	30	A, C	B, D	E-12 samples	A	E-11 samples	F, G
19	27	A, C	B, D	E-12 samples	DNS	E-11 samples	DNS
20	4	A, C	B, D	DNS	DNS	DNS	DNS
21	28	A, C	B, D	E-12 samples	A	E-11 samples	DNS
22	28	A, C	B, D	E-12 samples	A	E-11 samples	DNS
23	30	A, C	B, D	E-12 samples	A	E-11 samples	F, G
24	33	A, C	B, D	E-12 samples	A	E-14 samples	F, G
25	16	A, C	B, D	E-12 samples	DNS	DNS	DNS
26	30	A, C	B, D	E-12 samples	A	E-11 samples	F, G
27	33	A, C	B, D	E-12 samples	A	E-14 samples	F, G
28	30	A, C	B, D	DNS	A	E-11 samples	F, G
29	40	A, C	B, D	E-12 samples	A	E-20 samples	F, G, I
30	18	A, C	B, D	DNS	A	E-11 samples	F, G
31	31	A, C	B, D	E-12 samples	A	E-12 samples	F, G
32	28	A, C	B, D	E-12 samples	A	E-11 samples	DNS
33	33	A, C	B, D	E-12 samples	A	E-14 samples	F, G
34	18	A, C	B, D	DNS	A	E-11 samples	F, G
35	30	A, C	B, D	E-12 samples	A	E-11 samples	F, G
36	30	A, C	B, D	E-12 samples	A	E-11 samples	F, G
37	4	A, C	B, D	DNS	DNS	DNS	DNS
38	16	A, C	B, D	E-12 samples	DNS	DNS	DNS

A = NHU First-draw Sample
B = PF First-draw Sample
C = NHU 45-Second Flushed Sample
D = PF 45-Second Flushed Sample
E = Sequential Sample

F = 3-minute Flushed Sample
G = 5-minute Flushed Sample
H = 7-minute Flushed Sample
I = 10-minute Flushed Sample
DNS = Site did not sample

Table S3: Summary of samples collected at each site using each sampling protocol.

First-draw and 45-second flushed samples – Results for first-draw and 45-second flushed samples using the normal household use (NHU) and pre-flushed (PF) sampling protocols are presented in Table S4 below.

In addition to the first-draw samples, a 45-second flush sample was collected by running the water for 45 seconds immediately following the collection of the NHU first-draw and PF first-draw samples during the March/April sampling. Overall, the 45-second flush sample results were higher than the first-draw results, and yielded a higher percentage of results above the lead AL. A total of 32 NHU/45-second flushed and 32 PF/45-second flushed samples were collected, with 6 NHU 45-second flushed results above the lead AL (19%), and 5 PF/45-second flushed results above the AL (16%). The total number of 45-second flush sample results above the lead AL was 11 of 64 (17%); a percentage significantly higher than the first-draw results (2%).

First-draw and 45-second Flushed Sample Lead Results (µg/L)						
Site	A (Mar/Apr)	C (Mar/Apr)	B (Mar/Apr)	D (Mar/Apr)	B (June)	A (Sept/Oct)	B (Sept/Oct)
1	5.93	11.3	5.94	11.9	6.98	7.37	9.19
3	5.60	12.0	6.01	6.71	5.82	10.0	8.28
4	3.25	6.76	3.12	2.56	3.61	DNS	DNS
5	3.84	13.2	4.97	14.1	2.56	3.04	2.76
6	2.31	1.90	2.07	2.13	2.50	2.44	2.25
7	4.74	15.3	4.62	**24.9**	4.91	5.12	4.03
8	11.2	**32.2**	7.12	**28.0**	11.1	**17.5**	9.24
9	6.82	**15.9**	9.80	**17.7**	10.4	15.3	8.29
10	5.46	**25.0**	3.06	**21.6**	3.70	4.98	3.46
11	8.08	4.13	3.85	5.30	2.15	3.53	2.96
12	1.99	**17.2**	9.36	5.45	1.80	2.27	5.35
13	2.68	3.50	3.05	2.94	DNS	2.53	1.88
17	2.83	4.00	2.50	3.70	2.37	2.65	2.73
18	5.98	9.57	6.60	12.4	4.55	5.80	4.75
19	2.59	4.69	1.92	8.27	2.90	DNS	3.01
21	2.81	6.87	2.60	13.8	3.16	4.13	2.99
22	3.91	9.19	3.36	7.93	2.06	3.21	2.29
23	5.97	13.1	5.80	11.5	8.30	9.16	7.02
24	3.33	6.10	3.05	4.98	4.63	7.57	6.62
25	3.41	3.75	ND	ND	4.28	DNS	DNS
26	3.89	3.02	3.12	3.45	3.51	4.53	4.88
27	5.19	4.53	5.36	3.76	8.06	8.30	12.6
28	2.51	4.99	2.47	4.70	DNS	4.26	3.94
29	12.8	13.5	12.1	**28.6**	13.7	1.9	17.6
30	7.56	12.5	4.72	6.52	DNS	8.39	7.88
31	2.53	3.16	2.92	12.3	4.03	4.67	5.97
32	6.18	2.29	2.90	7.82	3.08	3.36	2.94
33	4.25	**16.4**	3.51	14.0	5.18	5.55	5.52
34	4.12	1.51	1.88	3.30	DNS	2.07	1.52
35	3.53	5.28	2.04	10.5	2.86	5.02	3.44
36	5.11	11.1	4.56	8.76	5.02	5.88	4.61
38	1.87	1.60	1.66	2.30	1.92	DNS	DNS
Ave	4.76	9.23	4.25	9.74	4.82	5.73	5.45
n	32	32	32	32	28	28	29

A = NHU First-draw Sample   D = PF 45-Second Flushed Sample
B = PF First-draw Sample   DNS = Site did not sample
C = NHU 45-Second Flushed Sample   n = number of samples collected

Table S4a: First-Draw and 45-Second Flushed Sampling Results. Samples that were above the lead AL are in bold, and samples that contained visible particulates are shaded yellow.

Summary of NHU and PF First-Draw Results					
	NHU (Mar/Apr)	PF (Mar/Apr)	PF (June)	NHU (Sept/Oct)	PF (Sept/Oct)
90th %ile Pb Value (µg/L)	8	7	8	10	9
No. of Samples	32	32	28	29	30
No. > AL	0	0	0	2	1

Table S4b: Comparison of LCR-equivalent 90th percentile results using alternative first-draw protocols.

Sequential sampling results (June 2011) – The sequential sampling approach provided a more reliable (volumetric) method for assessing corrosion as compared to a flushed (time-based) approach. Attempting to characterize the flow at each site would require an evaluation of the plumbing materials and dimensions, as well as the condition of the plumbing materials at each site, is not a feasible or reliable protocol for compliance monitoring.

The results of the each liter in the sequential sampling conducted in June are tabulated below in Table S5 by site.

June Sequential Sampling Results by Site/Liter (µg/L)												
	Liter											
Site	1	2	3	4	5	6	7	8	9	10	11	12
01	6.98	10.5	24.8	27.8	27.5	24.3	22.6	17.8	19.5	20.0	21.1	19.6
03	5.82	8.91	9.18	10.2	13.1	14.6	14.4	12.9	12.1	11.6	10.7	9.34
04	3.61	5.56		7.17	8.90	9.41	8.78	8.30	5.14	3.59	3.11	2.96
05	2.56	6.73	14.0	17.3	16.5	9.85	6.72	6.29	6.01	5.73	5.65	5.60
06	2.50	2.23	2.28	2.57	2.44	2.75	2.65	2.59	3.57	5.26	4.67	4.80
07	4.91	5.45	6.28	6.73	7.03	22.9	23.6	19.7	16.3	16.2	16.7	14.6
08	11.1	12.8	21.6	19.7	32.0	33.5	32.2	28.9	32.1	29.7	24.2	18.7
09	10.4	18.0	20.8	20.0	17.9	17.0	15.8	14.7	14.3	12.9	11.5	9.48
10	3.70	5.20	5.39	6.49	14.9	23.6	22.4	21.9	23.9	20.2	20.7	20.9
11	2.15	2.58	2.76	2.97	3.36	3.61	3.73	3.82	4.28	4.11	4.11	4.43
12	1.80	2.95	3.55	6.69	20.9	26.9	25.7	25.1	24.9	22.4	15.9	7.80
17	2.37	8.46	7.12	7.20	7.27	10.5	9.91	9.56	22.6	23.3	24.7	6.30
18	4.55	5.73	5.12	6.43	5.41	5.62	5.5	9.38	14.0	12.1	11.3	11.6
19	2.90	2.62	2.41	8.22	4.58	3.16	4.02	5.07	4.57	4.06	3.31	2.82
21	3.16	3.12	3.08	2.97	13.0	20.6	18.7	16.4	16.3	14.2	6.78	3.21
22	2.06	2.82	5.11	5.42	6.89	12.6	7.80	7.11	6.52	6.55	7.55	7.45
23	8.30	9.06	11.1	13.5	13.2	12.4	11.7	11.0	9.55	7.16	5.69	5.41
24	4.63	6.06	6.43	5.24	5.06	4.91	5.02	8.21	11.9	12.6	11.9	12.2
25	4.28	4.28	4.15	4.23	6.82	10.9	11.3	10.9	10.1	9.68	9.17	8.82
26	3.51	3.83	3.99	3.93	3.86	3.99	4.00	4.01	4.12	4.39	4.30	4.23
27	8.06	9.13	9.84	10.3	10.4	11.4	13.10	13.9	14.2	13.3	12.2	10.1
29	13.7	35.7	18.8	17.7	16.8	16.5	16.6	15.7	14.4	14.1	13.7	13.4
31	4.03	5.03	5.14	6.17	13.1	15.4	15.6	16.3	20.8	18.8	7.91	4.48

June Sequential Sampling Results by Site/Liter (µg/L)												
	Liter											
Site	1	2	3	4	5	6	7	8	9	10	11	12
32	3.08	2.29	2.07	2.28	6.95	15.5	9.91	9.27	8.30	6.12	2.60	1.65
33	5.18	6.85	10.0	7.74	9.61	13.9	**16.4**	13.5	12.3	13.7	10.7	9.95
35	2.86	7.89	12.9	11.9	9.85	8.59	7.28	6.82	6.23	5.34	5.02	4.83
36	5.02	6.90	7.68	8.46	9.90	9.81	9.51	9.34	9.19	8.93	9.20	9.19
38	1.92	3.04	3.06	3.04	2.91	3.03	3.12	3.07	3.36	3.21	3.04	3.76
Min	*1.80*	*2.23*	*2.07*	*2.28*	*2.44*	*2.75*	*2.65*	*2.59*	*3.36*	*3.11*	*2.60*	*1.65*
Max	*13.7*	*35.7*	*24.8*	*27.8*	*32.0*	*33.5*	*32.2*	*28.9*	*32.1*	*29.7*	*24.7*	*20.9*
Ave	*4.83*	*7.28*	*8.42*	*9.07*	*11.1*	*13.1*	*12.4*	*11.7*	*12.5*	*11.7*	*10.3*	*8.50*
90th %ile	*10.4*	*12.8*	*20.8*	*19.7*	*20.9*	*24.3*	*23.6*	*21.9*	*23.9*	*22.4*	*21.1*	*18.7*

Table S5: Summary of June Sequential Sampling Results. Samples that were above the lead AL are in bold, and samples that contained visible particulates are shaded yellow.

Sequential Sampling Results (September and October 2011) – The results of the each liter in the sequential sampling conducted in September and October are tabulated below in Table S6 by site. Considerably more sample results contained visible particulates than in previous rounds. The presence of particulates may be a result of the Chicago Fire Department exercising valves during the time period when samples were being collected.

All sites collected at least 11 sequential samples, and some sites with high sample results in June collected additional samples. The additional sequential sample results are included here but were not included in the data analyses, since extra samples were collected only from sites with high lead. A review of the data, including and excluding these additional results was performed to ensure that a bias has not been introduced, and the review indicates that the study findings are not significantly affected by including or excluding the data. With the additional 39 samples included, a total of 80 of 358 sample results (22%) exceeded the lead AL. Using only samples 1 through 11 from each site, a total of 75 of 319 sample results (24%) exceeded the lead AL. For the purpose of the data analyses, the first liter sample from the sequential samples in June and Sept/Oct also serve as the first-draw PF sample.

Sept/Oct Sequential Sampling Results by Site/Liter (µg/L)										
	Liter									
Site	1	2	3	4	5	6	7	8	9	10	11
01	9.19	12.8	**21.4**	**22.3**	**22.0**	**19.6**	**16.5**	**15.6**	14.5	14.2	13.8
03	8.28	5.58	5.17	6.43	8.46	14.9	**19.6**	**16.4**	**15.4**	14.3	**17.1**
05	2.76	10.8	12.2	10.9	12.3	7.21	5.49	5.24	4.65	5.30	5.40
06	2.25	2.18	3.43	2.37	2.30	2.28	2.81	2.32	2.20	4.16	5.03
07	4.03	4.27	5.74	5.75	9.87	15.1	15.3	15.2	12.1	14.8	13.9
08	9.24	8.95	9.45	11.8	**18.3**	**25.0**	**22.7**	**22.3**	**22.9**	**19.1**	**15.8**
09	8.29	**20.0**	**18.8**	**21.3**	**20.0**	**17.6**	**16.3**	**15.7**	14.6	14.8	**16.1**
10	3.46	6.27	6.23	5.05	14.8	**21.4**	**33.1**	**29.8**	**32.4**	**28.1**	**27.7**
11	2.96	4.05	3.90	3.91	4.30	4.44	4.35	4.71	5.02	4.75	4.47
12	5.35	**15.7**	**16.4**	**19.8**	**23.0**	**30.3**	**25.7**	**22.4**	**19.0**	**17.3**	12.2
13	1.88	7.73	9.01	3.57	2.53	3.85	2.96	2.17	2.85	7.55	5.74
17	2.73	2.38	5.45	4.41	4.07	4.09	3.72	3.42	3.35	3.42	3.17
18	4.75	5.09	4.91	5.53	4.81	8.17	8.61	8.67	11.6	11.6	11.4
19	3.01	3.07	2.75	3.80	3.25	3.37	5.80	6.01	6.15	5.18	3.83
21	2.99	3.35	3.03	3.04	**16.8**	**18.2**	**16.1**	13.2	14.9	15.0	5.24
22	2.29	2.86	5.60	5.39	6.32	8.49	7.42	7.20	6.64	7.09	7.36
23	7.02	8.00	8.99	11.0	12.5	12.1	12.8	11.8	10.5	12.1	10.1
24	6.62	8.84	7.30	6.38	6.45	6.59	6.82	10.6	14.5	13.2	12.8
26	4.88	4.61	4.52	4.46	4.52	4.26	5.18	5.40	5.94	5.72	5.82
27	12.6	12.4	12.2	12.5	12.5	13.1	**16.3**	**18.0**	**18.9**	**19.6**	**17.3**
28	3.94	5.58	5.39	5.32	5.39	5.11	5.73	5.65	5.30	5.49	5.55
29	**17.6**	**36.7**	**18.3**	**17.3**	**16.6**	**15.9**	**15.9**	14.3	**16.2**	12.8	13.2
30	7.88	7.46	8.67	9.54	9.09	11.0	12.9	**22.9**	**31.3**	**31.8**	**33.1**
31	5.97	5.82	5.20	6.72	15.6	13.4	**17.3**	**18.5**	**23.9**	**16.3**	5.70
32	2.94	2.24	2.03	2.22	5.50	**17.3**	9.42	9.07	8.63	7.64	3.50
33	5.52	6.26	12.8	9.09	12.0	14.1	**21.6**	**16.6**	**16.5**	**15.8**	14.1
34	1.52	1.72	1.69	1.62	1.73	2.66	2.91	2.87	3.17	2.10	1.90
35	3.44	7.42	14.6	**18.9**	**16.0**	12.5	10.1	9.56	7.60	8.18	7.21
36	4.61	5.01	5.51	6.11	13.0	11.6	10.3	10.4	10.9	10.3	9.93
Min	*1.52*	*1.72*	*1.69*	*1.62*	*1.73*	*2.28*	*2.81*	*2.17*	*2.20*	*2.10*	*1.90*
Max	*17.6*	*36.7*	*21.4*	*22.3*	*23.0*	*30.3*	*33.1*	*29.8*	*32.4*	*31.8*	*33.1*
Ave	*5.45*	*7.83*	*8.30*	*8.50*	*10.5*	*11.9*	*12.2*	*12.0*	*12.5*	*12.0*	*10.6*
90th %ile	*9.19*	*12.8*	*16.4*	*18.9*	*18.3*	*19.6*	*21.6*	*22.3*	*22.9*	*19.1*	*17.1*

Table S6a: Summary of September/October sequential sampling results used in data analyses. Samples that were above the lead AL are in bold, and samples that contained visible particulates are shaded yellow.

	Sept/Oct Sequential Sampling Results by Site/Liter (µg/L)								
	Liter								
Site	12	13	14	15	16	17	18	19	20
01	13.9	14.1	11.7	--	--	--	--	--	--
03	--	--	--	--	--	--	--	--	--
05	--	--	--	--	--	--	--	--	--
06	--	--	--	--	--	--	--	--	--
07	12.7	9.29	6.52	6.03	--	--	--	--	--
08	12.8	9.34	7.93	6.27	--	--	--	--	--
09	--	--	--	--	--	--	--	--	--
10	--	**27.1**	**21.1**	10.7	--	--	--	--	--
11	--	--	--	--	--	--	--	--	--
12	6.98	3.28	2.04	--	--	--	--	--	--
13	--	--	--	--	--	--	--	--	--
17	2.84	2.62	2.59	--	--	--	--	--	--
18	--	--	--	--	--	--	--	--	--
19	--	--	--	--	--	--	--	--	--
21	--	--	--	--	--	--	--	--	--
22	--	--	--	--	--	--	--	--	--
23	--	--	--	--	--	--	--	--	--
24	12.8	15.3	15.4	--	--	--	--	--	--
26	--	--	--	--	--	--	--	--	--
27	**16.0**	12.8	9.24	--	--	--	--	--	--
28	--	--	--	--	--	--	--	--	--
29	11.1	10.1	9.21	9.01	9.29	8.99	8.77	8.73	8.39
30	--	--	--	--	--	--	--	--	--
31	4.17	--	--	--	--	--	--	--	--
32	--	--	--	--	--	--	--	--	--
33	12.4	11.5	10.1	--	--	--	--	--	--
34	--	--	--	--	--	--	--	--	--
35	--	--	--	--	--	--	--	--	--
36	--	--	--	--	--	--	--	--	--
Min	2.84	2.62	2.04	6.03	9.29	8.99	8.77	8.73	8.39
Max	**16.0**	**27.1**	**21.1**	10.7	9.29	8.99	8.77	8.73	8.39
Ave	10.6	11.5	9.58	8.00	9.29	8.99	8.77	8.73	8.39
90th %ile	13.9	15.3	15.4	10.7	9.29	8.99	8.77	8.73	8.39

Table S6b: Summary of Supplemental September/October sequential sampling results not used in data analyses. Samples that were above the lead AL are in bold, and samples that contained visible particulates are shaded yellow.

Stagnation Times – Volunteers were asked to record the date and time water was last used, and the date and time when sampling began for each set of samples. Table S6c is a summary table which contains the stagnation times for the sequential samples, which is the amount of time the water sat motionless in the household prior to sample collection.

Sample Collection Stagnation Times			
June Sequential Sampling		Sept/Oct Sequential Sampling	
Site	Stagnation Time (hrs:mins)	Site	Stagnation Time (hrs:mins)
1	6:32	1	8:04
3	7:13	3	7:45
4	7:06	5	7:45
5	7:00	6	8:00
6	9:10	7	7:13
7	7:24	8	6:05
8	7:35	9	7:20
9	8:15	10	***
10	6:06	11	7:08
11	7:00	12	6:26
12	8:06	13	***
17	6:25	17	6:55
18	8:43	18	12:53
19	6:30	19	***
21	6:15	21	6:00
22	6:20	22	6:15
23	7:45	23	9:00
24	8:33	24	7:01
25	8:32	26	7:00
26	7:00	27	7:45
27	7:00	28	8:00
29	***	29	***
31	7:26	30	10:45
32	7:13	31	7:30
33	7:02	32	6:54
35	7:04	33	9:06
36	7:45	34	7:05
38	7:13	35	6:55
		36	8:47
***Volunteer did not record date/time the water was last used, but said it was the day before and was at least 6 hours before sampling.			

Table S6c: Summary of stagnation times for sequential sampling.

Seasonal Variability – Table S6d contains a site by site comparison of lead concentrations.

Seasonal Variability (Spring vs. Fall & Summer vs. Fall)					
First-Draw NHU	Sept/Oct > Mar/Apr	First-Draw PF	Sept/Oct > Mar/Apr	Sequential Samples	Sept/Oct > June
No. of Sample Pairs	28	No. of Sample Pairs	29	No. of Sample Pairs	285
No. Higher in Sept/Oct	19	No. Higher in Sept/Oct	20	No. Higher in Sept/Oct	156
% Higher in Sept/Oct	68%	% Higher in Sept/Oct	69%	% Higher in Sept/Oct	55%
First-Draw Samples: Mar/Apr vs. Sept/Oct (Same Site, Same First-Draw Protocol Compared)					
Sequential Samples: June vs. Sept/Oct (Same Site/Same Liter Compared)					

Table S6d: Seasonal variability effects observed.

Flushed sample results – The results of the flushed samples collected in September and October are tabulated in Table S7 by site. Most sites collected a 3 minute and 5 minute flushed sample. Some sites collected a 3, 5, and 7 minute flushed sample; and one site (site 29) collected a 3, 5, and 10 minute flushed sample, due to the length of the service line (159 ft / 48.5 m).

A flushed sample is collected by fully opening the sample tap and letting the water run for at least five minutes prior to a minimum 6 hour stagnation period. The date and time of the PF was recorded. After the minimum 6 hour stagnation period, and immediately before beginning the flushed sample collection, the date and time were again recorded and used as the start of sampling. The 3, 5, 7 and 10 minutes are measured from that start time, and water was not turned off between samples. For sequential sampling and flushed samples, the water was not turned off between samples.

EPA's current Public Notification Handbook includes instructions that advise residents to run the water between 30 and 45 seconds before collecting water for consumption if the water has not been used for an extended period of time. Running the water (flushing) for 45 seconds resulted in high lead levels at the tap for some sites. The flushed sampling results in this study indicate that EPA should develop a more appropriate flushing guidance, based on whether a home has a LSL or not, and the length of the LSL.

For homes with long LSLs, such as Site 29 (159 ft / 48.5 m), flushing may not be a practical way to reduce lead levels, as lead levels did not decline any further following 3, 5 and 10 minutes of flushing. In the case of site 29, residents would likely have a minimum of approximately 8 to 11 µg/L of lead in the drinking water for all water consumed, and should consider installing a water filter or using bottled water for drinking and cooking.

	Mar/Apr 2011	Mar/Apr 2011	Sept/Oct 2011	Sept/Oct 2011	Sept/Oct 2011	Sept/Oct 2011
Site	NHU 45sec	PF 45sec	3min	5min	7min	10min
01	11.3	11.9	6.48	6.56	6.97	
03	12.0	6.71	3.78	2.93		
04	6.76	2.56				
05	13.2	14.1				
06	1.90	2.13				
07	**15.3**	**24.9**	5.49	5.46	5.32	
08	**32.2**	**28.0**	8.25	5.54	5.71	
09	**15.9**	**17.7**	14.3	7.23		
10	**25.0**	**21.6**	4.95	4.30	4.09	
11	4.13	5.30	1.75	1.69		
12	**17.2**	5.45	1.78	1.45	1.33	
13	3.50	2.94				
17	4.00	3.70	2.88	2.76	2.86	
18	9.57	12.4	4.15	3.71		
19	4.69	8.27				
20	2.80	2.54				
21	6.87	13.8				
22	9.19	7.93				
23	13.1	11.5	5.64	4.54		
24	6.10	4.98	6.38	12.4		
25	3.75	ND				
26	3.02	3.45	5.06	3.23		
27	4.53	3.76	15.0	14.1		
28	4.99	4.70	4.82	3.26		
29	13.5	**28.6**	11.9	10.9		10.8
30	12.5	6.52	5.80	4.82		
31	3.16	12.3	3.78	3.76		
32	2.29	7.82				
33	**16.4**	14.0	4.40	4.06		
34	1.51	3.30	1.83	1.75		
35	5.28	10.5	5.53	4.03		
36	11.1	8.76	7.19	5.29		
38	1.60	2.30				

Flushed Sample Summary Table (μg/L)

NHU 45sec Samples were collected following the collection of the First-Draw NHU samples by running the water for 45 seconds following the collection of the First-Draw NHU sample.

PF 45sec Samples were collected following the collection of the First-Draw PF samples by running the water for 45 seconds following the collection of the First-Draw PF sample.

3min, 5min, 7min, and 10min flushed samples were collected after pre-flushing the tap for at least 5 minutes prior to the minimum 6 hour stagnation time during which no water was used in the home. Following the stagnation period and prior to sample collection, residents flushed the tap for 3 min to collect the 3min sample, and then an additional 2min for the 5min sample or 4min for the 7min sample. One site (site 29) had the longest lead service line so this site collected a 3 min, 5 min and 10min flushed sample (water was flushed for an additional 5 minutes following the collection of the 5min sample to collect the 10 min flushed sample). Water was not turned off in between samples to avoid the water hammer effect. Residents were instructed to have the bottles ready to insert under the faucet at the appropriate time.

Site 20 and Site 21 are the same residence. Site 20 was the upstairs bathroom and Site 21 was the kitchen sink. Note that neither the 45sec NHU nor PF samples from the upstairs tap captured any LSL water, while at least one of the kitchen tap samples did.

Table S7: Summary table of flushed sample results. Samples that were above the lead AL are in bold, and samples that contained visible particulates are shaded yellow.

Classification of Disturbed LSL Sites – A summary of the classification of each site as "disturbed", "undisturbed", or "indeterminate" is presented in Table S8, along with the number of samples collected per site and the number and percentage of sample results above the lead action level. The results from the "disturbed" and "undisturbed" sites are consistent with other research efforts showing that LSL disturbances result in higher lead levels[1-3].

Disturbed, Undisturbed and Indeterminate Site Summary								
Disturbed Sites	Total Samples Collected	# Samples Above AL (Disturbed)	Undisturbed Sites	Total Samples Collected	# Samples above AL (Undisturbed)	Indeterminate Sites	Total Samples Collected	# Samples above AL (Indeterminate)
01	27	16	03	27	4	12	27	17
05	27	2	04	14	0	21	27	7
07	27	11	06	27	0	33	27	6
08	27	19	11	27	0	---	---	---
09	27	15	13	15	0	---	---	---
10	27	15	18	27	0	---	---	---
17	27	3	19	27	0	---	---	---
27	27	5	22	27	0	---	---	---
28	15	0	23	27	0	---	---	---
29	27	15	24	27	0	---	---	---
30	15	4	25	14	0	---	---	---
31	27	10	26	27	0	---	---	---
35	27	2	32	27	2	---	---	---
---	---	---	34	15	0	---	---	---
---	---	---	36	27	0	---	---	---
---	---	---	38	16	0	---	---	---
Totals	327	117	Totals	371	6	Totals	81	30
% of samples above AL: 36%			% of samples above AL: 2%			% of samples above AL: 37%		

Table S8: Summary Table of Disturbed, Undisturbed and Indeterminate Sites, with the number and percentages of sample results above the lead AL for each site and each grouping.

Many direct LSL disturbances are localized to a specific segment of the LSL, and yet some sites have higher lead levels in sample liters over a significant portion of the LSL, not just in the immediate area of the LSL that was disturbed. A probable reason is that, except for the initial liter of water, each subsequent one-liter sample reflects both lead levels within the segment of the plumbing where the water stagnated as well as a contribution from the rest of the plumbing the water travelled through. For example, the fifth liter of water collected from a kitchen tap will not only capture the lead from the segment of LSL where the water stagnated, but it will also collect contributions from the plumbing downstream as the water passes through the remaining LSL and internal plumbing on the way to the kitchen tap. If the sample results only represented the portion of the plumbing where the water stagnated, it would be expected that a variety of metals would be found in the initial liters due to the presence of a variety of metallic plumbing materials and components, but only lead should be found in the LSL samples. In this study, a variety of metals was detected even in samples that represented LSL samples (Figure S6).

Specifically, for Site 9, information provided by the resident indicated that the internal pipe from the LSL to the kitchen tap was galvanized iron pipe. This was confirmed by the co-occurrence of higher levels of zinc and iron within the first liter of water in figure S6. There were no copper pipes in the home, so the presence of the copper is indicative of brass components (faucet, connectors, shut-off valve(s), and the water meter). Trace amounts of iron, zinc and copper are captured in the later liter samples as the water flows through the internal plumbing en route to the kitchen tap, along with traces of iron, potentially from the water main. It can reasonably be

assumed that the same phenomenon occurred for lead. Disturbed areas of the LSL have damaged scale, which can expose water passing through them to fresh lead. Therefore, lead measured in any sample upstream of the damaged area may include lead contributions from the damaged area.
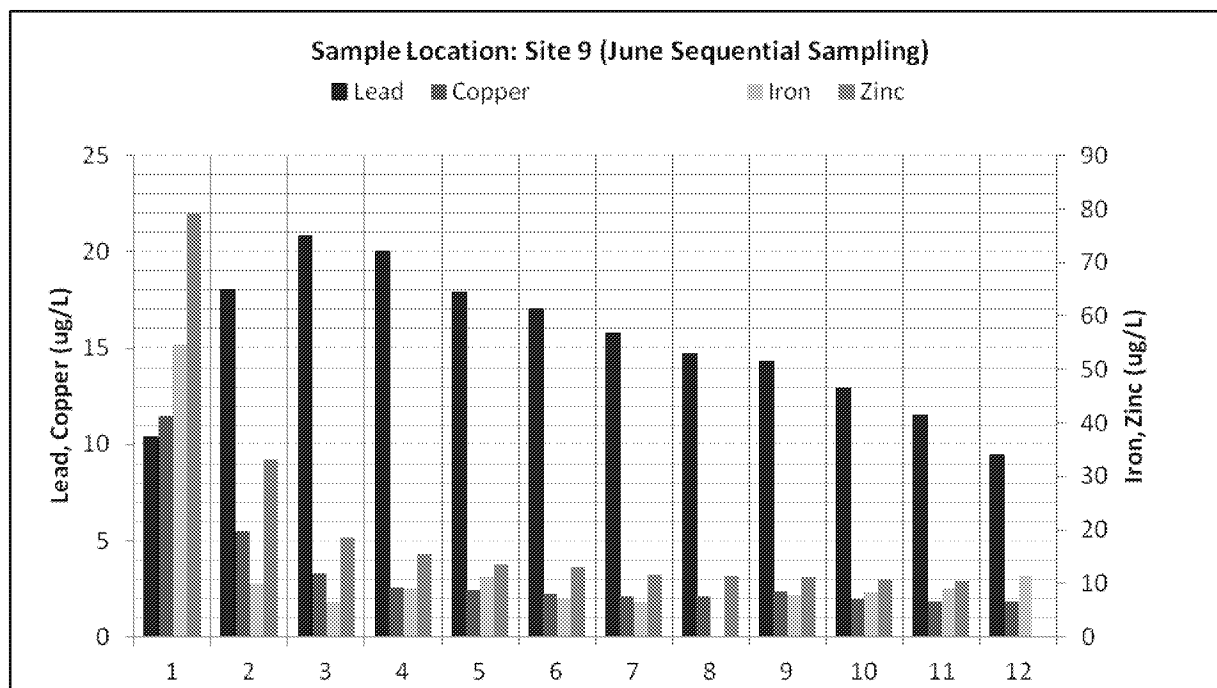
Figure S6: The LSL at Site 9 measures approximately 102 ft (31.1 m) from the water main to the meter. From the meter, there is approximately 13.5 ft (4.1 m) of 1 inch (2.54 cm) galvanized pipe to the kitchen tap.

Variability of lead levels in City B – A second city, City B, exceeded the lead AL during the July-Dec 2010 monitoring period, and was required to comply with the LSL replacement requirements in the LCR. Table S9 contains the compliance monitoring history for City B.

Monitoring Period Begin Date	Monitoring Period End Date	Number of Samples	Lead 90th Percentile Value (µg/l)
7/1/2011	12/31/2011	101	12
1/1/2011	6/30/2011	130	14
7/1/2010	12/31/2010	105	**23**
1/1/2009	12/31/2009	51	15
1/1/2008	12/31/2008	58	14
1/1/2007	12/31/2007	50	11
1/1/2006	12/31/2006	60	14
1/1/2005	12/31/2005	54	13
1/1/2004	6/30/2004	104	12
7/1/2003	12/31/2003	108	12
1/1/2002	12/31/2004	50	15
1/1/1999	12/31/1999	55	14
1/1/1998	12/31/1998	50	6
1/1/1997	12/31/1997	50	7
7/1/1996	12/31/1996	50	15
1/1/1996	6/30/1996	50	15
7/1/1992	12/31/1992	50	15
1/1/1992	6/30/1992	50	**21**

Table S9: City B 90th percentile compliance values (1992 – 2012). Samples that were above the lead AL are in bold.

S17 of 42

The sampling instructions presented in Figure S7 are in accordance with the LCR, and were used to collect the LSL samples in City B, which has approximately 25,000 LSLs.

Instructions for Lead Sample Collection

1 Make sure the faucet used for sample collection is _NOT_ attached to a water softener or any filtering device.
2 At bedtime, make sure the following rule is followed:
   o The water for the entire house, not just the faucet that is being used for collection, remains undisturbed for a period of _at least six hours_.
      ▪ No faucets in the house are used, which includes the bath tub, shower and sinks.
      ▪ The toilet is **not** flushed during this time period.
      ▪ The water is **not** run for an ice maker.
3 When you are ready to collect the sample:
   o Make sure the sample is taken before any other water is used.
      ▪ Open the collection container.
      ▪ Turn on the **cold** water.
      ▪ Allow the water to run until there is a significant change in temperature.
      ▪ Fill the container to the shoulder.
      ▪ Do **not** rinse the bottle out.
      ▪ _Immediately_ cap the sample container.
4 Fill out the enclosed chain of custody form and survey.
5 Fold and secure the chain of custody form and survey with a rubber band around the outside of the sample container.
   o Place the container outside where it was delivered.
❖ A city utilities employee will pick up the sample container. No one will enter your home. The sample must be left outside to be picked up.

Figure S7: LSL sampling instructions provided by City B to residents.

The sampling protocol used for collecting LSL samples ("allow the water to run until there is a significant change in temperature") can result in some sample results reflecting lead levels from internal plumbing rather than from within the LSLs.

The results from City B are presented below in Figure S8. Similar to the results presented for the study of Chicago, City B's results show significant variability in LSL lead levels across the system. Following the 2010 lead AL exceedance, the City B took 1,975 LSL samples, with a total of 1,762 results (89%) below the lead AL and 213 results (11%) above the lead AL. LSL results above the AL were significantly variable, ranging from 16 µg/L to 580 µg/L with a large number of sample results in exceedance of 50 µg/L.
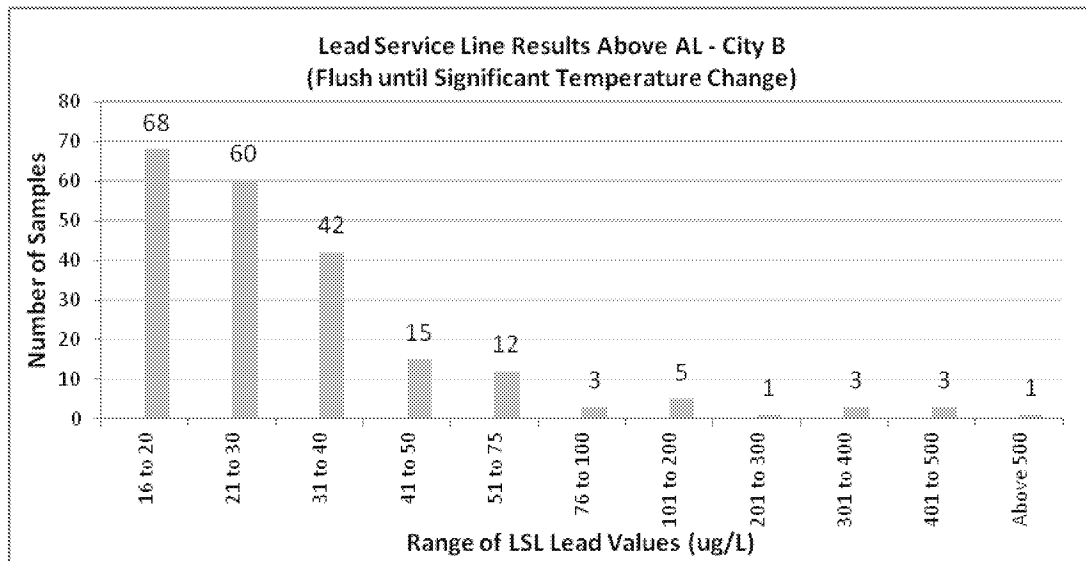
Figure S8: Range of lead values for City B LSL sampling results

Sequential Sampling Summary Graphs –The headers are color-coded based on whether the site has a disturbed LSL (red) or an undisturbed LSL (green). Sites for which this could not be determined (indeterminate sites) are color-coded orange. Water usage information is listed for each site. The samples which contained visible particulates are highlighted yellow, and the results that are above the lead AL are in bold text in the data tables. For sites that conducted sequential sampling in both June and Sept/Oct, the sequential sampling profiles were generally consistent during both sampling periods (see Figures S9 – S40).

Site 1		
Liter	June	Sept/Oct
1	7.0	9.2
2	11	13
3	25	21
4	28	22
5	28	22
6	24	20
7	23	17
8	18	16
9	20	15
10	20	14
11	21	14
12	20	14
13		14
14		12
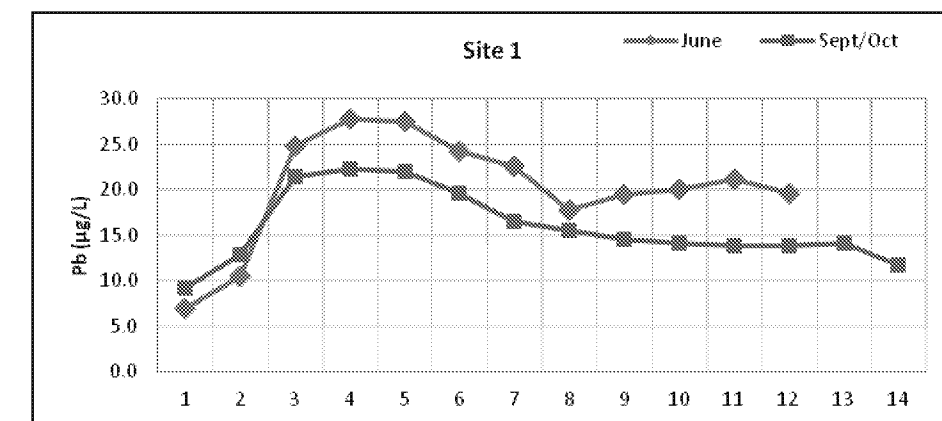
Disturbance(s): Water meter installed in 2010
Approximate LSL Length: 89 ft (27.1 m)
Ave Monthly Water Use: 3,444 gal. (13,037 L)

Figure S9: Sequential Lead Results - Sample Site #1 (June and Sept/Oct)

Site 3		
Liter	June	Sept/Oct
1	5.8	8.3
2	8.9	5.6
3	9.2	5.2
4	10	6.4
5	13	8.5
6	15	15
7	14	20
8	13	16
9	12	15
10	12	14
11	11	17
12	9.3	
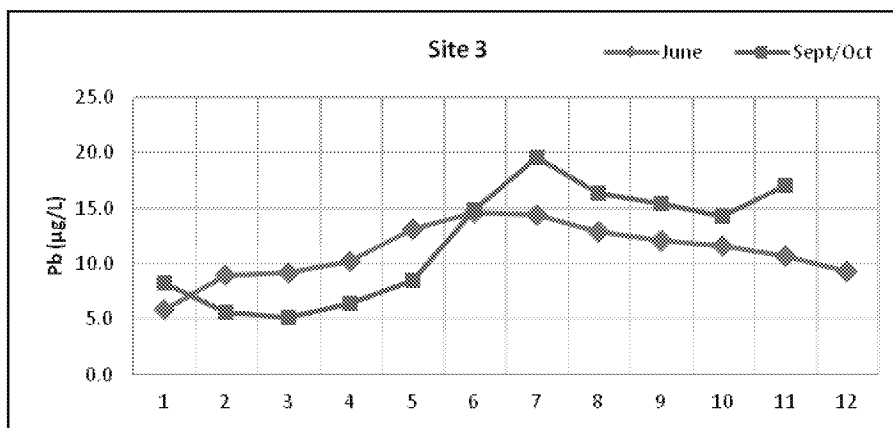
Disturbance(s): No known disturbance
Approximate LSL Length: 73 ft (22.3 m)
Ave Monthly Water Use: Not metered

Figure S10: Sequential Lead Results - Sample Site #3 (June and Sept/Oct)

Site 4	
Liter	June
1	3.61
2	5.56
3	
4	7.17
5	8.90
6	9.41
7	8.78
8	8.30
9	5.14
10	3.59
11	3.11
12	2.96
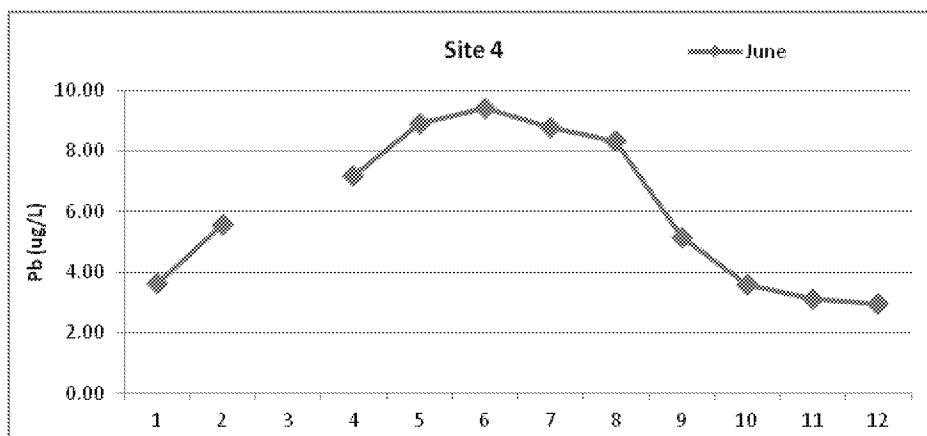
Disturbance(s): No known disturbance
Approximate LSL Length: Unknown
Ave Monthly Water Use: Not metered

Figure S11: Sequential Lead Results - Sample Site #4 (June)

Site 5		
Liter	June	Sept/Oct
1	2.6	2.8
2	6.7	11
3	14	12
4	17	11
5	17	12
6	9.9	7.2
7	6.7	5.5
8	6.3	5.2
9	6.0	4.7
10	5.7	5.3
11	5.7	5.4
12	5.6	
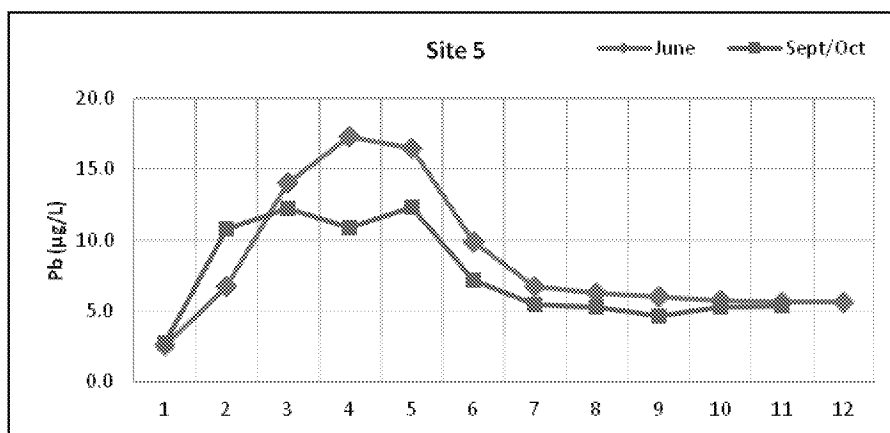
Disturbance(s): Water meter installed in 2011
Approximate LSL Length: 80 ft (24.4 m)
Ave Monthly Water Use: 10,400 gal. (39,368 L)

Figure S12: Sequential Lead Results - Sample Site #5 (June and Sept/Oct)

Site 6		
Liter	June	Sept/Oct
1	2.5	2.3
2	2.2	2.2
3	2.3	3.4
4	2.6	2.4
5	2.4	2.3
6	2.8	2.3
7	2.7	2.8
8	2.6	2.3
9	3.6	2.2
10	5.3	4.2
11	4.7	5.0
12	4.8	
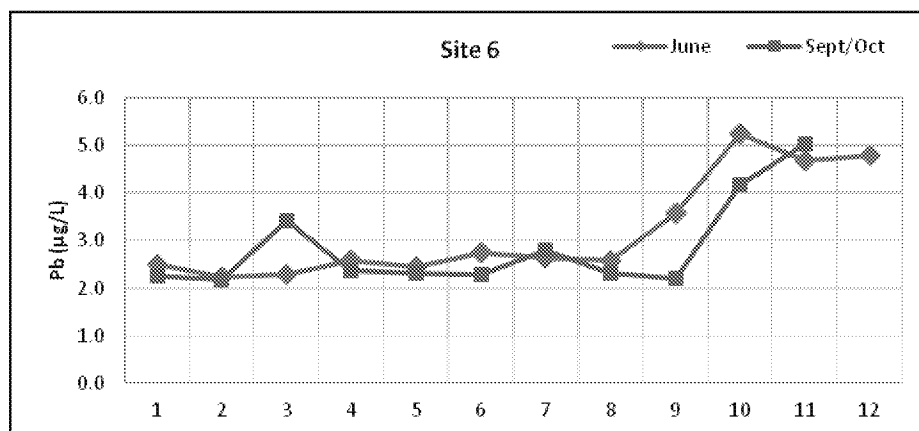
Disturbance(s): No known disturbance
Approximate LSL Length: 60 ft (18.3 m)
Ave Monthly Water Use: Not metered

Figure S13: Sequential Lead Results - Sample Site #6 (June and Sept/Oct)

Site 7		
Liter	June	Sept/Oct
1	4.9	4.0
2	5.5	4.3
3	6.3	5.7
4	6.7	5.8
5	7.0	9.9
6	23	15
7	24	15
8	20	15
9	16	12
10	16	15
11	17	14
12	15	13
13		9.3
14		6.5
15		6.0
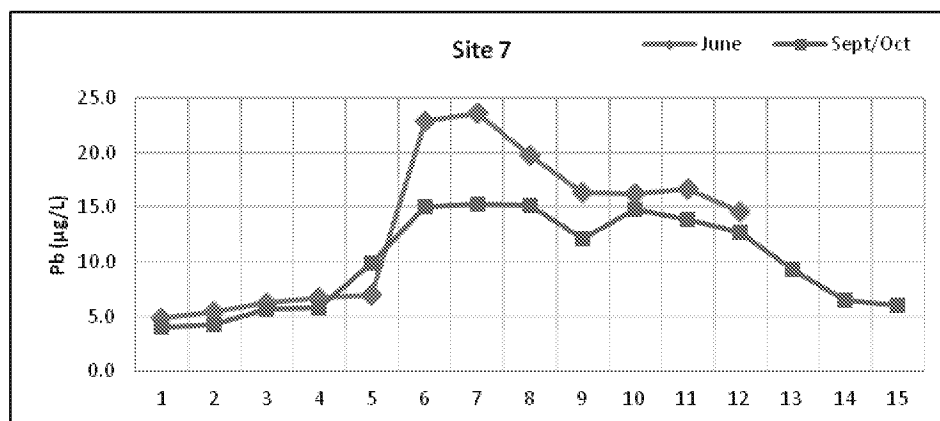
Disturbance(s): Street excavation, potential installation of Cu whip at service connection in 2008
Approximate LSL Length: 59+ ft (18.0+ m)
Ave Monthly Water Use: Not metered

Figure S14: Sequential Lead Results - Sample Site #7 (June and Sept/Oct)

Site 8		
Liter	June	Sept/Oct
1	11	9.2
2	13	9.0
3	22	10
4	20	12
5	32	18
6	34	25
7	32	23
8	29	22
9	32	23
10	30	19
11	24	16
12	19	13
13		9.3
14		7.9
15		6.3

Disturbance(s): Leak in parkway, repaired roundway in 2005.
Approximate LSL Length: 57 ft (17.4 m)
Ave Monthly Water Use: Not metered

Figure S15: Sequential Lead Results - Sample Site #8 (June and Sept/Oct)

Site 9		
Liter	June	Sept/Oct
1	10	8.3
2	18	20
3	21	19
4	20	21
5	18	20
6	17	18
7	16	16
8	15	16
9	14	15
10	13	15
11	12	16
12	10	
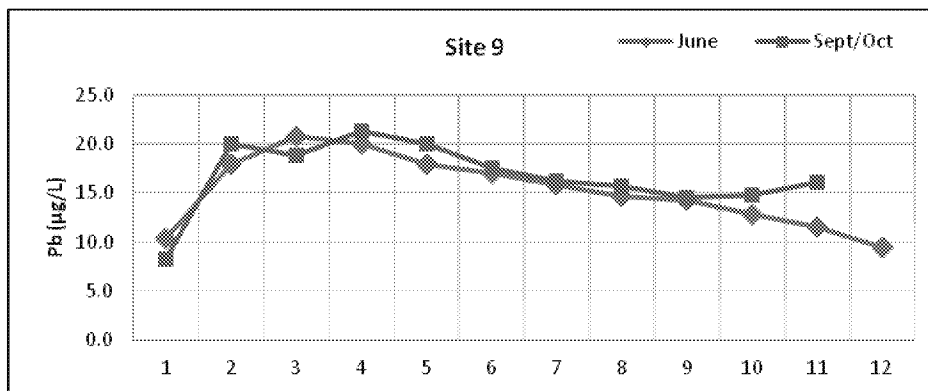
Disturbance(s): Water meter installed in 2008.
Approximate LSL Length: 102 ft (31.1 m)
Ave Monthly Water Use: 3,190 (12,075 L) – In Sept 2011, usage was 24,000 gal. (90,850 L) due to hose left running for one or more days. In calculating the overall average, the Sept 2010 value of 8,000 gal. (30,283 L) was also used for Sept 2011 instead of the 24,000 gal. (90,850 L) value.

Figure S16: Sequential Lead Results - Sample Site #9 (June and Sept/Oct)

Site 10		
Liter	June	Sept/Oct
1	3.7	3.5
2	5.2	6.3
3	5.4	6.2
4	6.5	5.1
5	15	15
6	24	21
7	22	33
8	22	30
9	24	32
10	20	28
11	21	28
12	21	
13		27
14		21
15		11
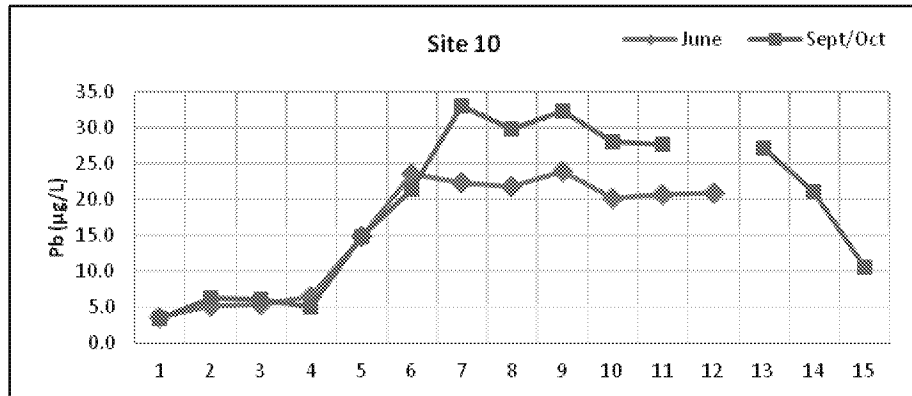
Disturbance(s): Service leak repair, water meter installed in 2009.
Approximate LSL Length: 48+ ft (14.6 m)
Ave Monthly Water Use: 1,826 gal. (6,912 L)

Figure S17: Sequential Lead Results - Sample Site #10 (June and Sept/Oct)

Site 11		
Liter	June	Sept/Oct
1	2.2	3.0
2	2.6	4.1
3	2.8	3.9
4	3.0	3.9
5	3.4	4.3
6	3.6	4.4
7	3.7	4.4
8	3.8	4.7
9	4.3	5.0
10	4.1	4.8
11	4.1	4.5
12	4.4	
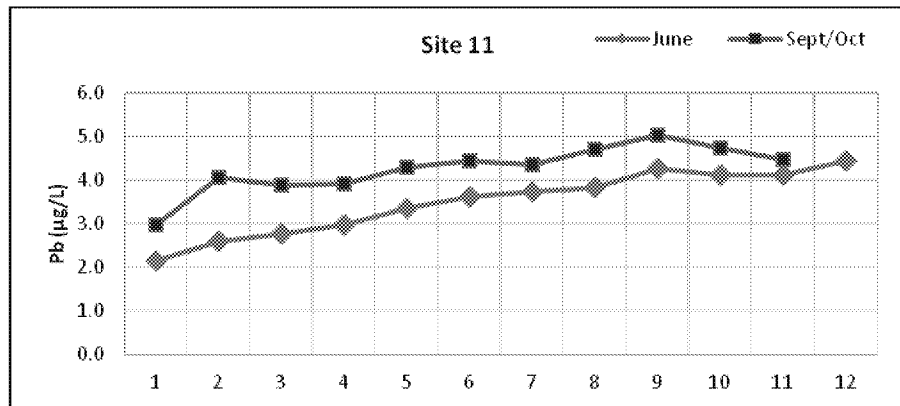
Disturbance(s): No known disturbance
Approximate LSL Length: 50 ft (15.2 m)
Ave Monthly Water Use: Not metered

Figure S18: Sequential Lead Results - Sample Site #11 (June and Sept/Oct)

Site 12		
Liter	June	Sept/Oct
1	1.8	5.4
2	3.0	16
3	3.6	16
4	6.7	20
5	21	23
6	27	30
7	26	26
8	25	22
9	25	19
10	22	17
11	16	12
12	7.8	7.0
13		3.3
14		2.0

Disturbance(s): Indeterminate
Approximate LSL Length: 53 (16.2 m)
Ave Monthly Water Use: Not metered

Figure S19: Sequential Lead Results - Sample Site #12 (June and Sept/Oct)

Site 13	
Liter	Sept/Oct
1	1.9
2	7.7
3	9.0
4	3.6
5	2.5
6	3.9
7	3.0
8	2.2
9	2.9
10	7.6
11	5.7
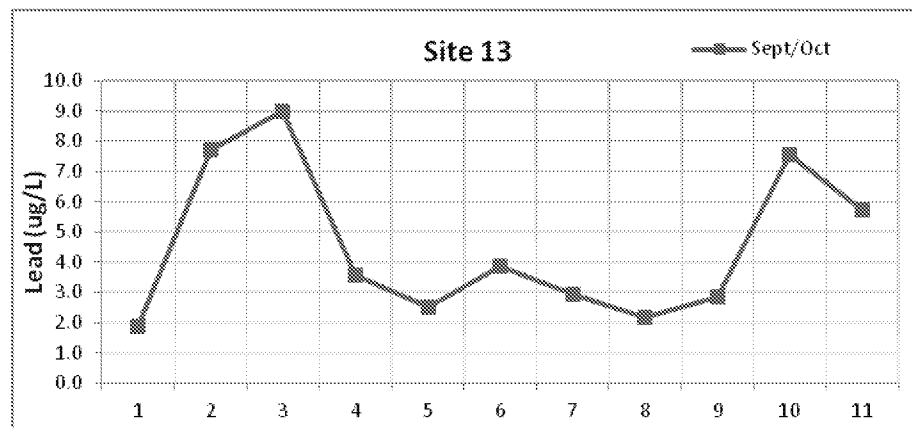
Disturbance(s): No known disturbance
Approximate LSL Length: 49+ ft (4.9 m)
Ave Monthly Water Use: Not metered

Figure S20: Sequential Lead Results - Sample Site #13 (Sept/Oct)

Site 17		
Liter	June	Sept/Oct
1	2.4	2.7
2	8.5	2.4
3	7.1	5.5
4	7.2	4.4
5	7.3	4.1
6	11	4.1
7	9.9	3.7
8	9.6	3.4
9	**23**	3.4
10	**23**	3.4
11	**25**	3.2
12	6.3	2.8
13		2.6
14		2.6
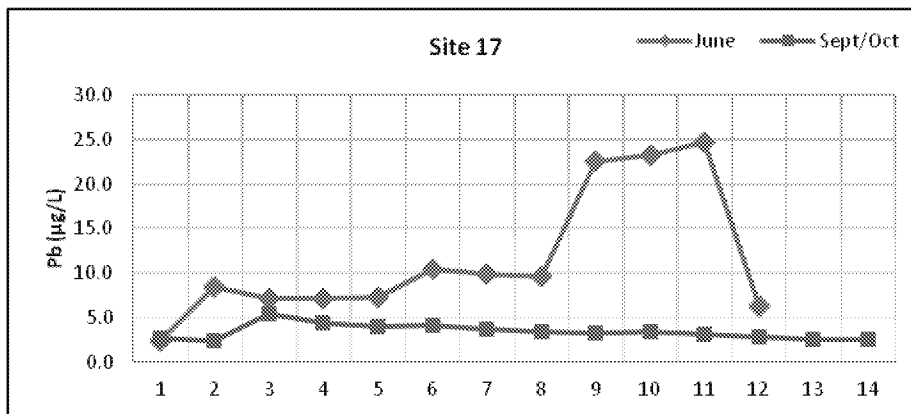
Disturbance(s): Meter replacement in 2008.
Approximate LSL Length: 58+ ft (17.7+ m)
Ave Monthly Water Use: 9,772 gal. (36,991 m)

Figure S21: Sequential Lead Results - Sample Site #17 (June and Sept/Oct)

Site 18		
Liter	June	Sept/Oct
1	4.6	4.8
2	5.7	5.1
3	5.1	4.9
4	6.4	5.5
5	5.4	4.8
6	5.6	8.2
7	5.5	8.6
8	9.4	8.7
9	14	12
10	12	12
11	11	11
12	12	
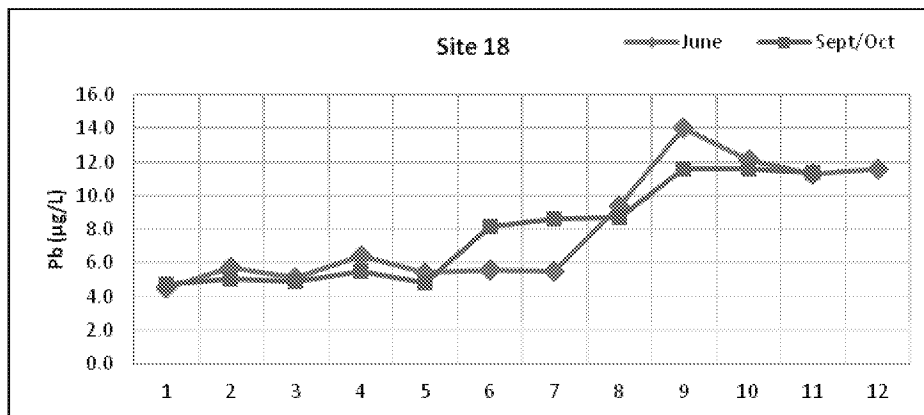
Disturbance(s): No known disturbance
Approximate LSL Length: 76 ft (23.2 m)
Ave Monthly Water Use: Not metered

Figure S22: Sequential Lead Results - Sample Site #18 (June and Sept/Oct)

Site 19		
Liter	June	Sept/Oct
1	2.9	3.0
2	2.6	3.1
3	2.4	2.8
4	8.2	3.8
5	4.6	3.3
6	3.2	3.4
7	4.0	5.8
8	5.1	6.0
9	4.6	6.2
10	4.1	5.2
11	3.3	3.8
12	2.8	
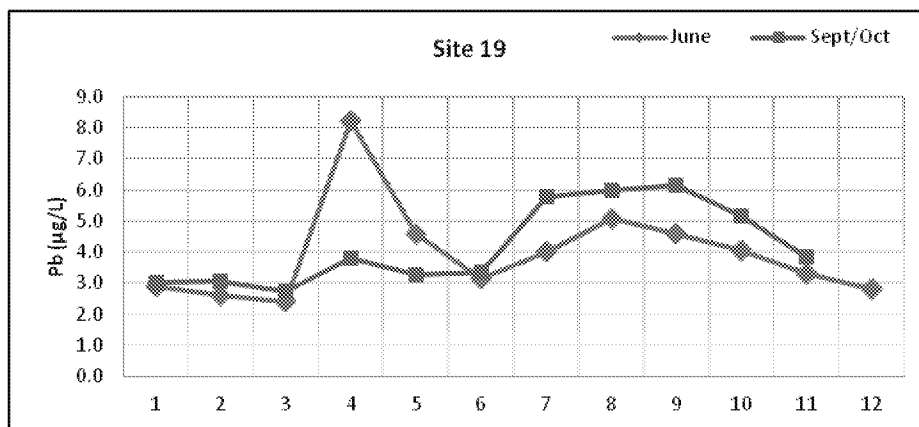
Disturbance(s): No known disturbance
Approximate LSL Length: 63 ft (19.2 m)
Ave Monthly Water Use: Not metered

Figure S23: Sequential Lead Results - Sample Site #19 (June and Sept/Oct)

Site 21		
Liter	June	Sept/Oct
1	3.2	3.0
2	3.1	3.4
3	3.1	3.0
4	3.0	3.0
5	13	17
6	21	18
7	19	16
8	16	13
9	16	15
10	14	15
11	7.0	5.2
12	3.2	
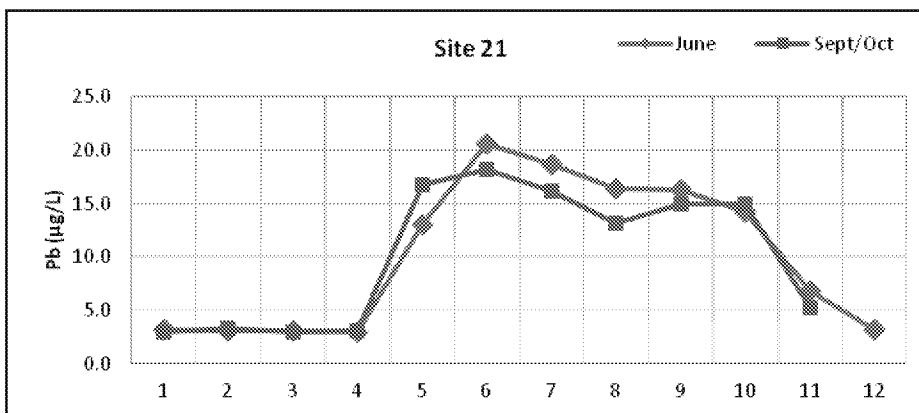
Disturbance(s): Indeterminate
Approximate LSL Length: 46 ft (14.0 m)
Ave Monthly Water Use: Not metered

Figure S24: Sequential Lead Results - Sample Site #21 (June and Sept/Oct)

Site 22		
Liter	June	Sept/Oct
1	2.1	2.3
2	2.8	2.9
3	5.1	5.6
4	5.4	5.4
5	6.9	6.3
6	13	8.5
7	7.8	7.4
8	7.1	7.2
9	6.5	6.6
10	6.6	7.1
11	7.6	7.4
12	7.5	
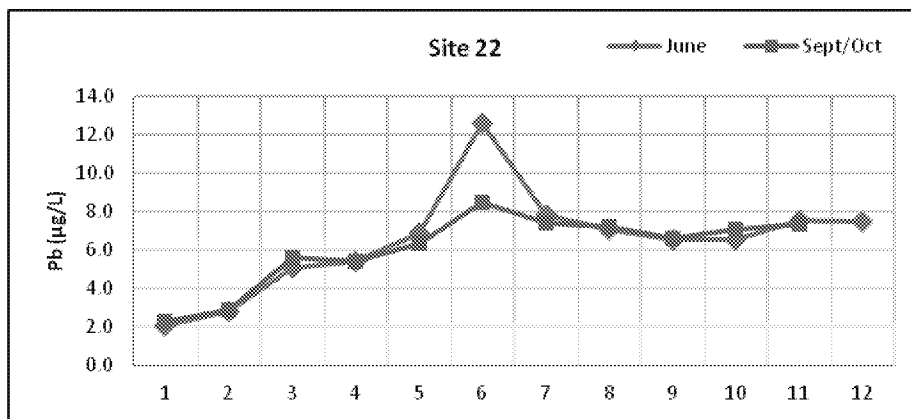
Disturbance(s): No known disturbance
Approximate LSL Length: 65 ft (19.8 m)
Ave Monthly Water Use: Not metered

Figure S25: Sequential Lead Results - Sample Site #22 (June and Sept/Oct)

Site 23		
Liter	June	Sept/Oct
1	8.3	7.0
2	9.1	8.0
3	11	9.0
4	14	11
5	13	13
6	12	12
7	12	13
8	11	12
9	9.6	11
10	7.2	12
11	5.7	10
12	5.4	
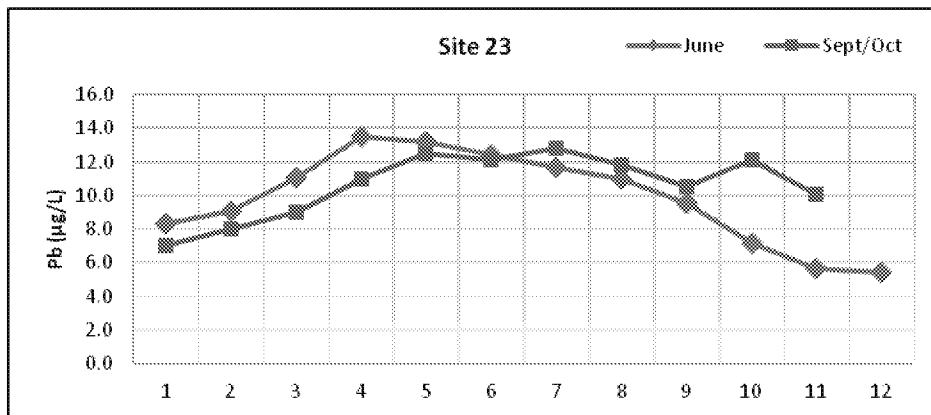
Disturbance(s): No known disturbance
Approximate LSL Length: 66 ft (20.1 m)
Ave Monthly Water Use: Not metered

Figure S26: Sequential Lead Results - Sample Site #23 (June and Sept/Oct)

Site 24		
Liter	June	Sept/Oct
1	4.6	6.6
2	6.1	8.8
3	6.4	7.3
4	5.2	6.4
5	5.1	6.5
6	4.9	6.6
7	5.0	6.8
8	8.2	11
9	12	15
10	13	13
11	12	13
12	12	13
13		15
14		15
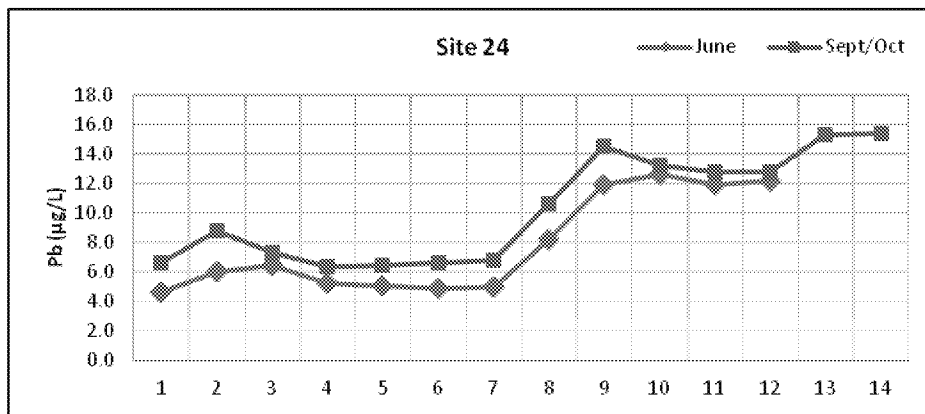
Disturbance(s): No known disturbance
Approximate LSL Length: 56 ft (17.1 m)
Ave Monthly Water Use: Not metered

Figure S27: Sequential Lead Results - Sample Site #24 (June and Sept/Oct)

Site 25		
Liter	June	Sept/Oct
1	3.5	4.9
2	3.8	4.6
3	4.0	4.5
4	3.9	4.5
5	3.9	4.5
6	4.0	4.3
7	4.0	5.2
8	4.0	5.4
9	4.1	5.9
10	4.4	5.7
11	4.3	5.8
12	4.2	
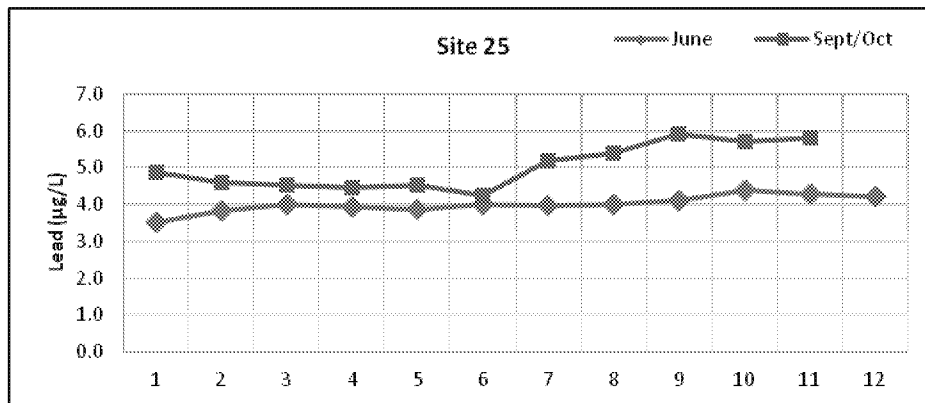
Disturbance(s): No known disturbance
Approximate LSL Length: 70 ft (21.3 m)
Ave Monthly Water Use: Not metered

Figure S28: Sequential Lead Results - Sample Site #25 (June and Sept/Oct)

Site 26		
Liter	June	Sept/Oct
1	3.5	4.9
2	3.8	4.6
3	4.0	4.5
4	3.9	4.5
5	3.9	4.5
6	4.0	4.3
7	4.0	5.2
8	4.0	5.4
9	4.1	5.9
10	4.4	5.7
11	4.3	5.8
12	4.2	
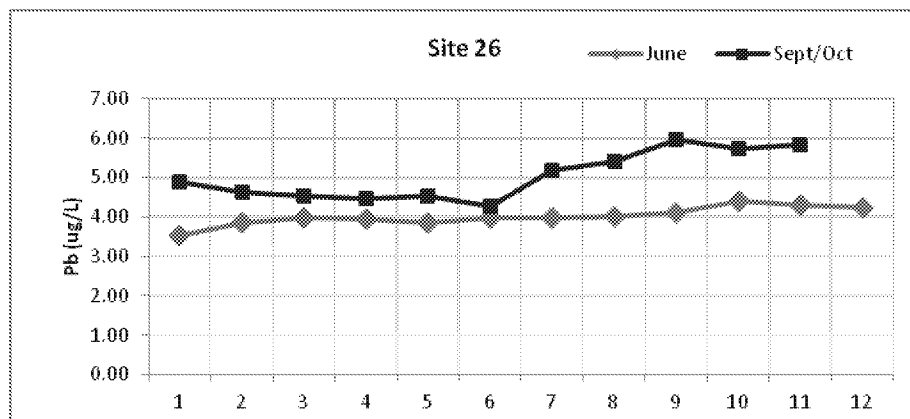
Disturbance(s): No known disturbance
Approximate LSL Length: 66 ft (20.1 m)
Ave Monthly Water Use: Not metered

Figure S29: Sequential Lead Results - Sample Site #26 (June and Sept/Oct)

Site 27		
Liter	June	Sept/Oct
1	8.1	13
2	9.1	12
3	9.8	12
4	10	13
5	10	13
6	11	13
7	13	16
8	14	18
9	14	19
10	13	20
11	12	17
12	10	16
13		13
14		9.2
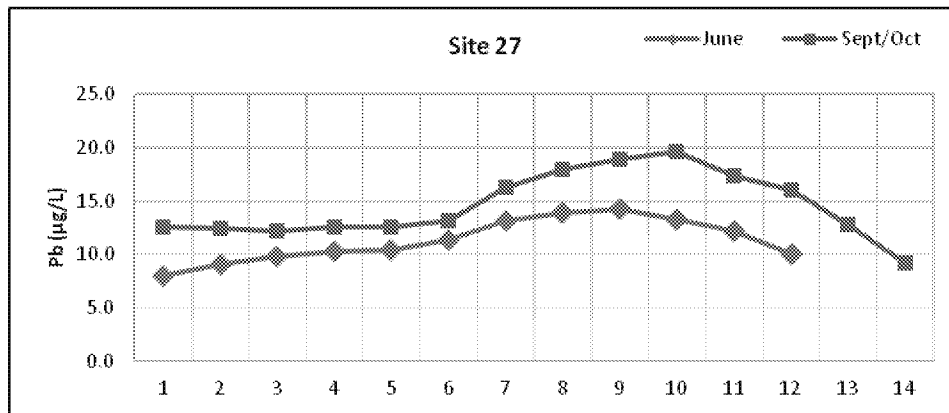
Disturbance(s): Meter replacement in 2010.
Approximate LSL Length: 47+ ft (14.3 m)
Ave Monthly Water Use: 4267 gal. (16,152 L)

Figure S30: Sequential Lead Results - Sample Site #27 (June and Sept/Oct)

Site 28	
Liter	Sept/Oct
1	3.9
2	5.6
3	5.4
4	5.3
5	5.4
6	5.1
7	5.7
8	5.7
9	5.3
10	5.5
11	5.6
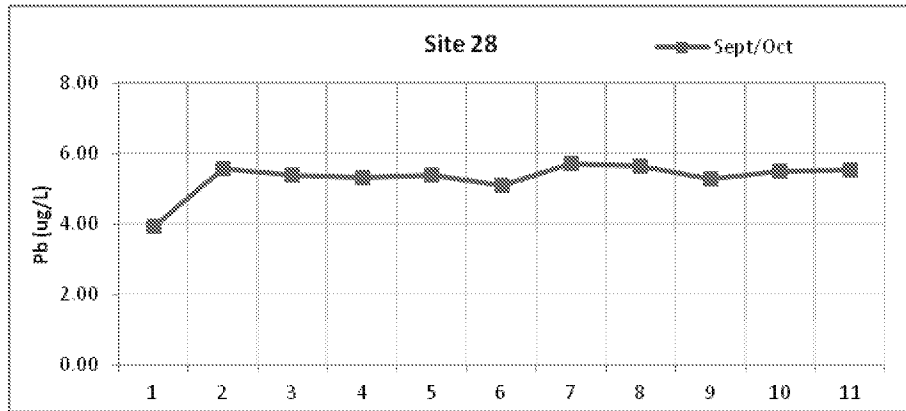
Disturbance(s): Meter replacement in 2009.
Approximate LSL Length: 61+ ft (18.6+ m)
Ave Monthly Water Use: 4273 gal. (16,175 L)

Figure S31: Sequential Lead Results - Sample Site #28 (Sept/Oct)

Site 29		
Liter	June	Sept/Oct
1	14	18
2	36	37
3	19	18
4	18	17
5	17	17
6	17	16
7	17	16
8	16	14
9	14	16
10	14	13
11	14	13
12	13	11
13		10
14		9.2
15		9.0
16		9.3
17		9.0
18		8.8
19		8.7
20		8.4
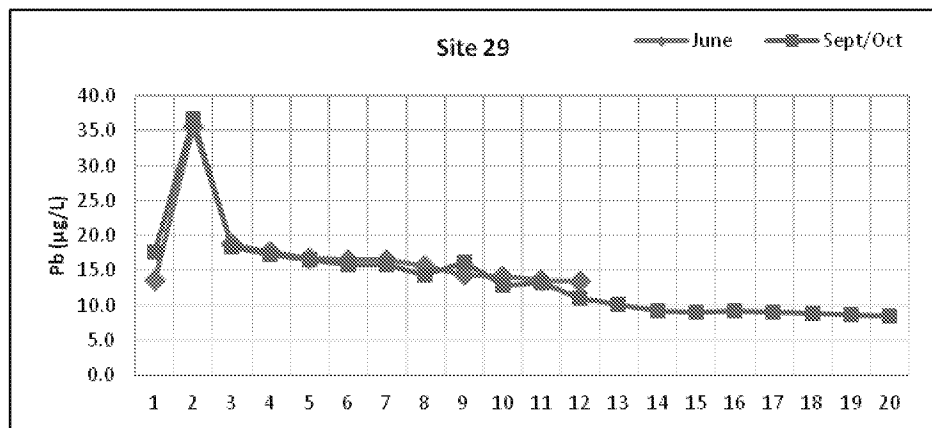
Disturbance(s): Probable Approximate LSL leak repair, meter installed in 2010.
Approximate LSL Length: 159 ft (48.5 m)
Ave Monthly Water Use: 1,438 gal. (5,443 L)

Figure S32: Sequential Lead Results - Sample Site #29 (June and Sept/Oct)

S31 of 42

Liter	Sept/Oct
1	7.9
2	7.5
3	8.7
4	9.5
5	9.1
6	11
7	13
8	23
9	31
10	32
11	33
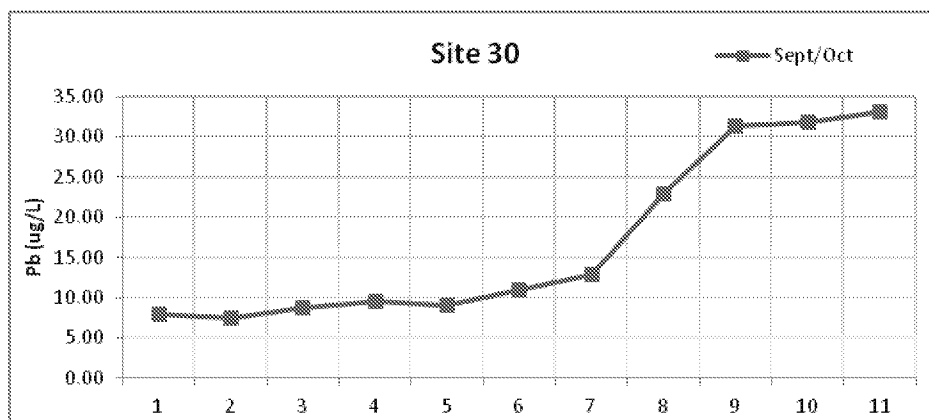
Disturbance(s): Broken water main in 2000, sidewalk replaced & street re-surfacing.
Approximate LSL Length: 49+ ft (14.9 m)
Ave Monthly Water Use: Not metered

Figure S33: Sequential Lead Results - Sample Site #30 (Sept/Oct)

Liter	June	Sept/Oct
1	4.0	6.0
2	5.0	5.8
3	5.1	5.2
4	6.2	6.7
5	13	16
6	15	13
7	16	17
8	16	19
9	21	24
10	19	16
11	8	5.7
12	4.5	4.2
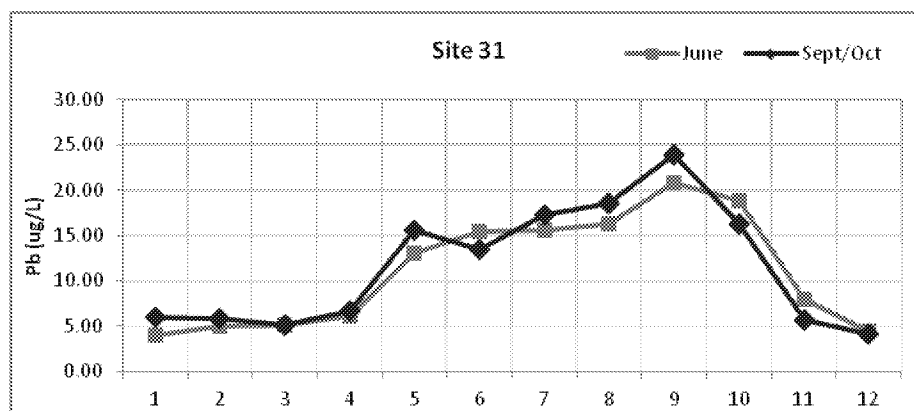
Disturbance(s): Approximate LSL leak repair in 2010.
Approximate LSL Length: 71+ ft (21.6+ m)
Ave Monthly Water Use: Not metered

Figure S34: Sequential Lead Results - Sample Site #31 (June and Sept/Oct)

Site 32		
Liter	June	Sept/Oct
1	3.1	2.9
2	2.3	2.2
3	2.1	2.0
4	2.3	2.2
5	7.0	5.5
6	16	17
7	9.9	9.4
8	9.3	9.1
9	8.3	8.6
10	6.1	7.6
11	2.6	3.5
12	1.7	
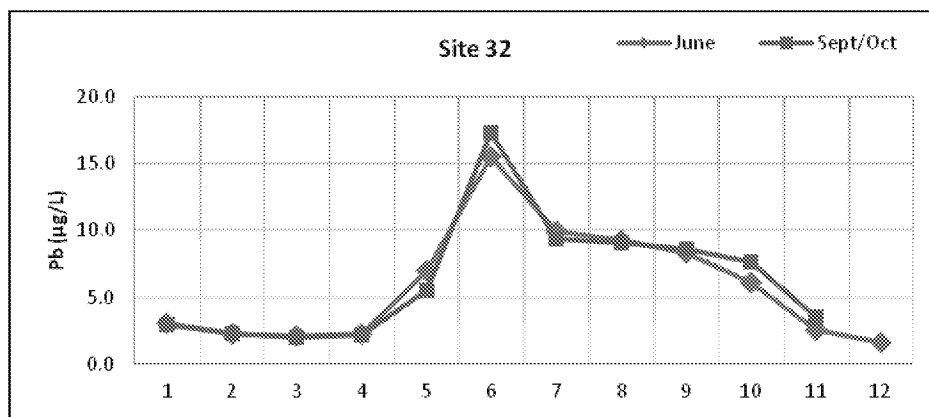
Disturbance(s): No known disturbance
Approximate LSL Length: 43 ft (13.1 m)
Ave Monthly Water Use: Not metered

Figure S35: Sequential Lead Results - Sample Site #32 (June and Sept/Oct)

Site 33		
Liter	June	Sept/Oct
1	5.2	5.5
2	6.9	6.3
3	10	13
4	7.7	9.1
5	9.6	12
6	14	14
7	16	22
8	14	17
9	12	17
10	14	16
11	11	14
12	10	12
11		12
12		10
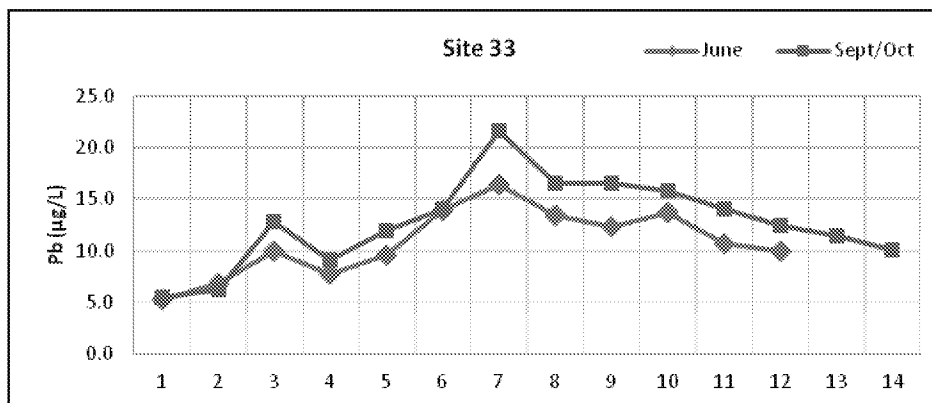
Disturbance(s): Indeterminate
Approximate LSL Length: 43+ ft (13.1 m)
Ave Monthly Water Use: Not metered

Figure S36: Sequential Lead Results - Sample Site #33 (June and Sept/Oct)

Site 34	
Liter	Sept/Oct
1	1.5
2	1.7
3	1.7
4	1.6
5	1.7
6	2.7
7	2.9
8	2.9
9	3.2
10	2.1
11	1.9
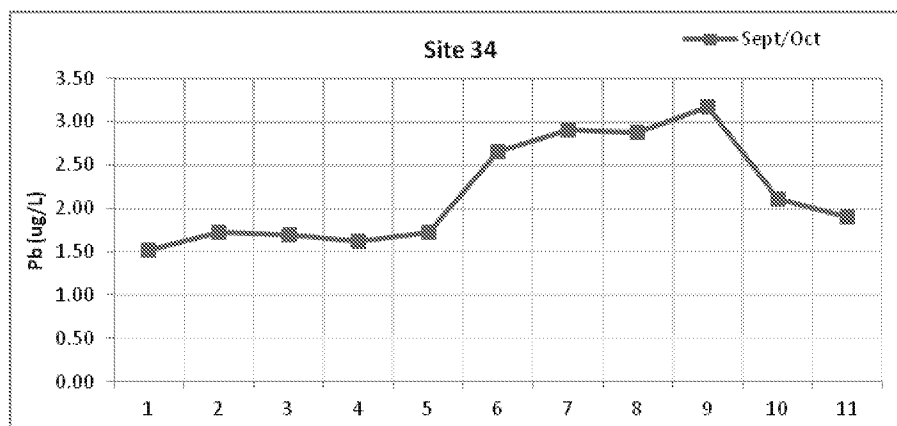
Disturbance(s): No known disturbance
Approximate LSL Length: Unknown
Ave Monthly Water Use: Not metered

Figure S37: Sequential Lead Results - Sample Site #34 (Sept/Oct)

Liter	June	Sept/Oct
1	2.9	3.4
2	7.9	7.4
3	13	15
4	12	**19**
5	9.9	**16**
6	8.6	13
7	7.3	10
8	6.8	9.6
9	6.2	7.6
10	5.3	8.2
11	5.0	7.2
12	4.8	
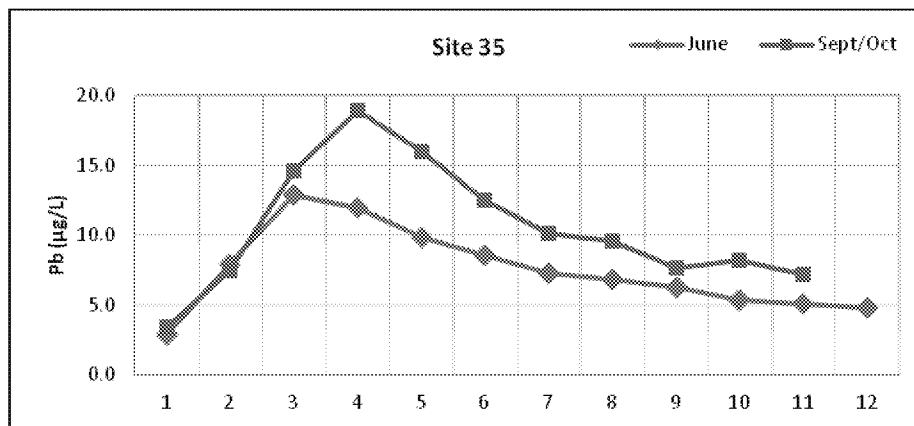
Disturbance(s): Meter installed in Aug 2011 (between June and Sept/Oct sampling).
Approximate LSL Length: 80 ft (24.4 m)
Ave Monthly Water Use: 4,667 gal. (17,667 L) – Data available only for Aug-Oct 2011

Figure S38: Sequential Lead Results - Sample Site #35 (June and Sept/Oct)

Site 36		
Liter	June	Sept/Oct
1	5.0	4.6
2	6.9	5.0
3	7.7	5.5
4	8.5	6.1
5	9.9	13
6	9.8	12
7	9.5	10
8	9.3	10
9	9.2	11
10	8.9	10
11	9.2	9.9
12	9.2	
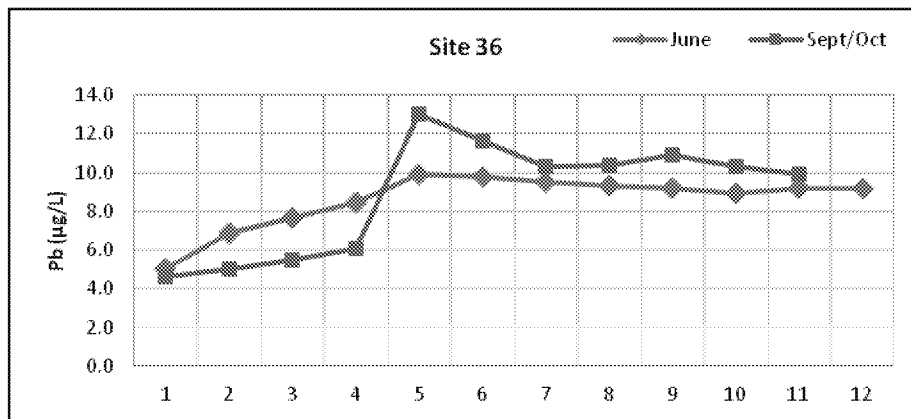
Disturbance(s): No known disturbance
Approximate LSL Length: 83+ ft (25.3 m)
Ave Monthly Water Use: Not metered

Figure S39: Sequential Lead Results - Sample Site #36 (June and Sept/Oct)

Site 38	
Liter	June
1	1.9
2	3.0
3	3.1
4	3.0
5	2.9
6	3.0
7	3.1
8	3.1
9	3.4
10	3.2
11	3.0
12	3.8
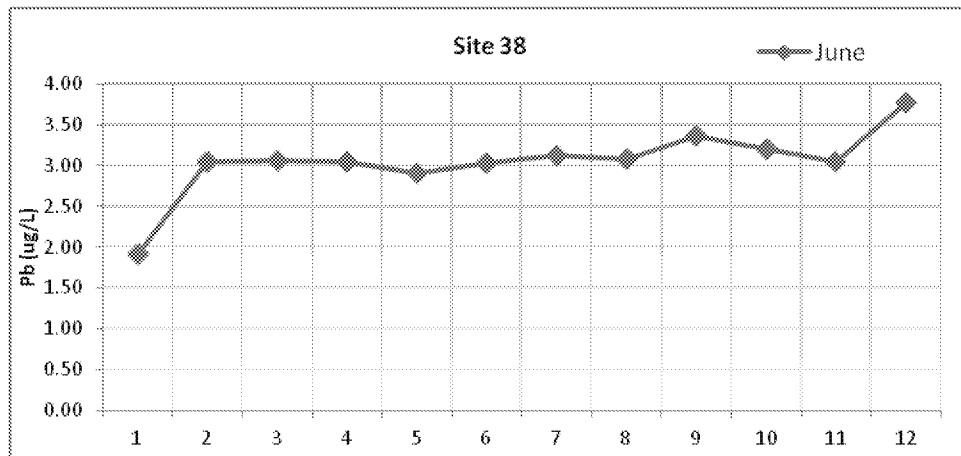
Disturbance(s): No known disturbance
Approximate LSL Length: 51 ft (15.5 m)
Ave Monthly Water Use: Not metered

Figure S40: Sequential Lead Results - Sample Site #38 (June)

Sampling collection and reporting instructions and forms

March/April sampling – The sampling instructions and forms below were used in the March/April sampling. Sampling was scheduled to conclude in March, but the sampling ran into April. As a result of the instructions below, some volunteers sampled one day at the kitchen tap and one day at the bathroom tap. The intent was to have all samples collected from the same tap, so volunteers that split the samples were asked to collect replacement samples so that a complete set of four samples was collected at the same tap. We chose the kitchen tap, and all samples collected thereafter were also collected at the kitchen tap. In addition, the 45-second flushed

sampling protocol was not used after the March/April sampling due to the complication with corroded galvanized pipe.

General Sampling Instructions
You will be taking a total of 8 samples for this study. One set of 4 samples will be taken in March 2011 and one set of 4 samples (using the same instructions) will be taken in August 2011. General Instructions for all four samples of a set *Sample #1 and Sample #2 must be collected one after another on the same day.* *Sample #3 and Sample #4 must also be collected one after another on the same day, and within the same week as Sample #1 and Sample #2.* All samples should be collected from taps that are generally used by your household for drinking water. <u>Do not collect samples from a taps that have not been used within the last 24 hours.</u> Use a kitchen or bathroom cold-water faucet for your sampling. <u>Do not collect samples from a tap that has a water filter or is connected to a water softener.</u> If you have a water softener or water filter on your kitchen tap, collect your sample from a bathroom tap that is not attached to the water softener or water filter, if possible.

Instructions for Collecting Sample #1

Important: Please make sure you use the bottle labeled 'Sample #1' for your first sample!

Collecting Sample #1: The first sample is to be collected after water throughout the household *has not been used for a minimum of 6 hours* (example: midnight to 6am). During these 6 hours, do not flush toilets, shower, or run water from other faucets. The best time to collect samples is either:
 1) First thing in the morning, before any water is used in the household; or 2) Immediately upon returning from work, and prior to using any water, as long as water has not been used in the household during the day.

1. When you are ready to collect your first sample, use the sample bottle labeled 'Sample #1'.
2. Do not run any water from the tap before collecting the first sample.
3. Place the opened sample bottle below the faucet and gently open the cold water tap.
4. Fill the sample bottle as you would normally fill a glass of water for drinking, up to the neck of the bottle (see photographs below) and turn off the water. Tightly cap the sample bottle.

Fill the bottle up to here
Do not overfill

Instructions for Collecting Sample #2

Important: Please make sure you use the bottle labeled 'Sample #2' for your second sample!

Collecting Sample #2: This sample is to be collected from the same faucet as Sample #1, immediately after collecting Sample #1.

1. Immediately after collecting Sample #1, <u>run the water for 45 seconds</u>. Shut off the water, and place the opened

sample bottle (labeled Sample #2) below the faucet and gently open the cold water tap.
2. Fill the sample bottle as you would normally fill a glass of water for drinking, up to the neck of the bottle (see photographs on first page) and turn off the water. Tightly cap the sample bottle.

Instructions for Collecting Sample #3

Important: Please make sure you use the bottle labeled 'Sample #3' for your third sample!

Collecting Sample #3: Collect on a different day in the same week as Samples #1 & #2.

1. *Before* letting the water sit for a minimum of 6 hours, run the water from the faucet for 5 minutes at a high rate, and then do not use any water in the household for at least 6 hours after that (Example: Run the water for 5 minutes at midnight before going to bed, and then do not use any water in the household until collecting the third sample at 6 am the following morning).
2. Do not run any more water from the tap before collecting the third sample. Place the opened sample bottle below the faucet and gently open the cold water tap.
3. Fill the sample bottle as you would normally fill a glass of water for drinking, up to the neck of the bottle (see photographs on first page) and turn off the water. Tightly cap the sample bottle.

Instructions for Collecting Sample #4

Important: Please make sure you use the bottle labeled 'Sample #4' for your fourth sample!

Collecting Sample #4: This sample is to be collected from the same faucet as Sample #3.

1. Immediately after collecting Sample #3, run the water for 45 seconds. Shut off the water, and place the opened sample bottle (labeled Sample #4) below the faucet and gently open the cold water tap.
2. Fill the sample bottle as you would normally fill a glass of water for drinking, up to the neck of the bottle (see photographs on first page) and turn off the water. Tightly cap the sample bottle.

Figure S41: March/April sampling instructions.

Sample Collection and Reporting Page

Sample Reporting – Sample #1	EPA Use: Visible Particulate? Yes ☐ No ☐

Sample ID (from Sample Bottle #1): _____   Date/time Sample #1 was collected: _____

Volunteer ID: _____   Sampling Location: Kitchen Faucet ☐   Bathroom Faucet ☐

Date/time the water was last used in household before collecting Sample #1: _____

Was sample #1 collected from a faucet that has a water softener or water filter?   Yes ☐   No ☐

Sample Reporting – Sample #2	EPA Use: Visible Particulate? Yes ☐ No ☐

Sample ID (from Sample Bottle #2): _____   Date/time Sample #2 was collected: _____

Volunteer ID: _____   Sampling Location: Kitchen Faucet ☐   Bathroom Faucet ☐

Date/time the water was last used in household before collecting Sample #2: _____

Was Sample #2 collected from the same faucet as Sample #1:   Yes ☐   No ☐

Sample Reporting – Sample #3	EPA Use: Visible Particulate? Yes ☐ No ☐

Sample ID (from Sample Bottle #3): _____   Date/time Sample #3 was collected: _____

Volunteer ID: _____   Sampling Location: Kitchen Faucet ☐   Bathroom Faucet ☐

Date/time the faucet was flushed before collecting Sample #3: _____

Was sample #3 collected from a faucet that has a water softener or water filter?   Yes ☐   No ☐

Sample Reporting – Sample #4	EPA Use: Visible Particulate? Yes ☐ No ☐

Sample ID (from Sample Bottle #4): _____   Date/time Sample #4 was collected: _____

Volunteer ID: _____   Sampling Location: Kitchen Faucet ☐   Bathroom Faucet ☐

Date/time the faucet was flushed before collecting Sample #4: _____

Was Sample #4 collected from the same faucet as Sample #3:   Yes ☐   No ☐

Have there been any plumbing repairs or plumbing work done within the household during the last six months (including installation of new faucets)?   Yes ☐   No ☐

If yes, explain briefly (*Example – 'New faucet installed one week ago'*):

FOR EPA USE: Samples received by _____   Date/Time: _____

Samples transferred to Region 5 Laboratory by _____   Date/Time: _____

Volunteer Certification: I have read the sampling instructions and have collected the samples in accordance with the instructions provided.

_____   OR _____
Signature/Date   *Volunteer ID/Date*

Figure S42: March/April sample collection and reporting form.

Sequential Sampling Instructions for June – The sampling instructions and forms below were used in the June sequential sampling.

Sequential Sampling Instructions

Please read all instructions before beginning your sampling

General Information
•Use <u>only the kitchen faucet</u> for all of these samples.

•Use only **cold water** and open the cold water tap all the way when filling the bottles.

•Fill each bottle to the top of the label on the sample bottle.

Sampling Instructions
•The night before sampling (right before everyone goes to bed) run the water from the kitchen tap for at least 5 minutes. Write down the date/time you finished running the water on the form on the back side of this page.

•The water must sit motionless in the home plumbing for at least 6 hours before collecting the samples so do not use water in the home after you finished running the water and until all samples are collected the following morning. Showering, flushing toilets, or other water use will affect the sampling results. It may help to tape a sign in the kitchen and bathrooms with a reminder not to use the water, in case people forget.

• The bottles are numbered, and <u>it is very important to collect them in order</u> (Sample 1 first, Sample 2 second, etc.).

•In the morning, when you are ready to sample, place the open bottles in order by sample number. You will be collecting the samples without shutting off the water in between samples, so you should remove the caps from all bottles so that you have all of the bottles ready to fill. You can put the caps on after all samples have been collected. Try not to let any water spill in between samples.

•Write down the date/time right before you sample on the form on the back side of this page.

•Begin by placing the Sample 1 bottle under the faucet and open the **cold water** slowly until the faucet is <u>fully open</u>. While one bottle is filling, grab the next bottle so that you are ready to move it under the faucet quickly.

•Once the bottle is filled to the top of the label, quickly place the Sample 2 bottle under the faucet, and continue until you have filled all sample bottles.

Sequential Sampling – Sample Collection and Reporting Form

Volunteer ID: _____

Sampling Information

Date/time the water was last used in household (the night before collecting the samples): _____

Date/Time Volunteer Began Collecting Samples: _____

Were All Samples Collected from the Kitchen Tap? Yes ☐ No ☐

FOR EPA USE: Samples received by _____ Date/Time: _____

Samples transferred to Region 5 Laboratory by _____ Date/Time: _____

EPA Use: Visible Particulate in any samples? Yes ☐ No ☐   If Yes – List Samples With Particulate _____

Volunteer Certification: I have read the sampling instructions and have collected the samples in accordance with the instructions provided.

_____ *OR* _____
Signature/Date   *Volunteer ID/Date*

Figure S43: June sampling instructions and sample collection and reporting form.

Sampling instructions for September/October – In the final round of sampling, the number and type of samples was customized to each site and sites collected 3 days of sampling. The instructions below were for a site collecting one NHU First-draw sample, 11 sequential samples and a 2 flushed samples. Some sites collected additional sequential samples and some collected 3 flushed samples instead of two.

Sampling Instructions
Please read all instructions before you start sampling.

General Information
  ✓ Use **only the kitchen faucet** for all of these samples.
  ✓ Use **only cold water**.
  ✓ Open the cold water tap all the way when filling the bottles.
  ✓ Fill each bottle to the top of the label on the sample bottle.

Sampling Instructions
  ✓ There are three different sets of samples for you to collect (Sample Set #1, #2 and #3).
  ✓ Each set will be taken on a different day. (The three sampling sets do not have to be taken on three days in a row.)
  ✓ A section of the reporting form (attached) needs to be filled in for each day of sampling.

A) Sample Set #1 (1 bottle, Blue Label) 1. The water must sit motionless in the home plumbing for at least 6 hours before collecting the sample. Typically, the night before taking the sample, make sure that no one uses water in the home until you collect the sample from the kitchen the following morning. 2. In the morning, when you are ready to sample, write down the date/time on the attached form. 3. Fill up the bottle with the BLUE LABEL. That's it for collecting the first sample set.
B) Sample Set #2 "Sequential Sampling" (11 bottles, WHITE LABELS) 1. The night before sampling (right before everyone goes to bed) run the water from the kitchen tap for at least 5 minutes. Write down the date/time you finished running the water on the form. After running the water for 5 minutes, it should sit motionless in the home plumbing for at least 6 hours. 2. In the morning, your first water usage should be collecting eleven samples in a row (one after another). Use the bottles with the WHITE LABELS. The samples should be collected without shutting off the water in between samples. To do this, remove the caps from all eleven bottles before you turn on the water. 3. Place the eleven open bottles in order by sample number before you start collecting the samples Try not to waste water in between the samples. You can put the caps on after all 11 samples have been collected. The bottles are numbered Seq 01, to Seq 11. **It is very important to collect the samples in order** (Seq 01 first, Seq 02 second, etc.). 4. Use the attached reporting form to note the date and time that you started taking the sample set.
C) Sample Set #3 (2 Bottles, GREEN LABEL and YELLOW LABEL) 1. The night before sampling (right before everyone goes to bed) run the water from the kitchen tap for at least 5 minutes. Write down the date/time you finished running the water on the form. After running the water for 5 minutes, it should sit motionless in the home plumbing for at least 6 hours. 2. In the morning, when you are ready to sample, write down the date/time on the attached reporting form. 3. Run the water for 3 minutes, then collect a sample in the jar with the GREEN LABEL. Continue to let the water run for an additional 2 minutes (for a total of 5 minutes), and collect the final sample in the bottle with the YELLOW LABEL.

Figure S44: Sept/Oct sampling instructions.

Sample Collection and Reporting – Sampling set # 1 (Blue label)

Volunteer ID: _____

Sampling Information

Date/time the water was last used in household (the night before collecting the samples): _____

Date/Time Volunteer Began Collecting Samples: _____

Were All Samples Collected from the Kitchen Tap? Yes ☐ No ☐

FOR EPA USE: Samples received by _____ Date/Time: _____

Samples transferred to Region 5 Laboratory by _____ Date/Time: _____

EPA Use: Visible Particulate in any samples? Yes ☐ No ☐ If Yes – List Samples With Particulate
_____

Sample Collection and Reporting - Sampling set # 2 (11 samples, White labels)

Volunteer ID: _____

Sampling Information

Date/time the water was last used in household (the night before collecting the samples): _____

Date/Time Volunteer Began Collecting Samples: _____

Were All Samples Collected from the Kitchen Tap? Yes ☐ No ☐

FOR EPA USE: Samples received by _____ Date/Time: _____

Samples transferred to Region 5 Laboratory by _____ Date/Time: _____

EPA Use: Visible Particulate in any samples? Yes ☐ No ☐ If Yes – List Samples With Particulate
_____

Sample Collection and Reporting - Sampling set # 3 (Green and Yellow labels)

Volunteer ID: _____

Sampling Information

Date/time the water was last used in household (the night before collecting the samples): _____

Date/Time Volunteer Began Collecting Samples: _____

Were All Samples Collected from the Kitchen Tap? Yes ☐ No ☐

FOR EPA USE: Samples received by _____ Date/Time: _____

Samples transferred to Region 5 Laboratory by _____ Date/Time: _____

EPA Use: Visible Particulate in any samples? Yes ☐ No ☐ If Yes – List Samples With Particulate
_____

Volunteer Certification: I have read the sampling instructions and have collected the samples in accordance with the instructions provided.

_____ *OR* _____

Signature/Date   *Volunteer ID/Date*

Figure S45: Sept/Oct sample collection and reporting form.

Literature Cited/References

1.   Triantafyllidou, S.; Edwards, M., Galvanic corrosion after simulated small-scale partial lead service line replacements. *Journal American Water Works Association* **2011**, *103*, (9), 85-+.
2.   Renner, R., Reaction to the Solution: Lead Exposure Following Partial Service Line Replacement. *Environmental health perspectives* **2010**, *118*, (5).
3.   Cartier, C.; Arnold Jr, R. B.; Triantafyllidou, S.; Prévost, M.; Edwards, M., Effect of Flow Rate and Lead/Copper Pipe Sequence on Lead Release from Service Lines. *Water Research* **2012**, *46*, (13), 4142-4152.

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION 5

IN THE MATTER OF:)
)
Alternative Lead in Drinking Water)   VARIANCE UNDER
Reduction Treatment Technique   )   SECTION 1415(A)(3) OF
for Wisconsin Public Water Systems)   SDWA
)
)

INTRODUCTION

1. Statutory and Regulatory Background

Under the Safe Drinking Water Act, 42 U.S.C. '' 300f-300j-26 (SDWA), U.S. EPA promulgates national primary drinking water regulations (NPDWRs), which specify for certain drinking water contaminants either a maximum level or treatment technique with which public water systems (PWSs) must comply. U.S. EPA has promulgated an NPDWR for lead and copper, the lead and copper rule (LCR), 40 C.F.R. Part 141, Subpart I, that consists of a treatment technique requiring PWSs to take various steps to ensure that users of their system are not exposed to levels of lead and/or copper in drinking water that would result in adverse health effects. The LCR requires all Community Water Systems (CWSs) and Non-Transient Non-Community Water Systems (NTNCWSs) to optimize corrosion control and to conduct tap water monitoring to ensure that lead and copper levels are minimized at users= taps. If tap water levels exceed either Aaction level@ (AL) of 0.015 mg/L for lead or 1.3 mg/L for copper, in more than 10 percent of drinking water tap samples (i.e., exceeds the AL as a 90th percentile value), PWSs are required to take additional steps, including delivering public education materials to users about the health risks of lead in drinking water (for lead AL exceedances), treating source water if it contains elevated lead and/or copper levels, or installing corrosion control treatment (CCT). For systems that continue to exceed the lead AL after optimizing CCT, the system must begin replacing at least seven percent of lead service lines (LSLs) in the system per year. LSLs that contribute less than 0.015 mg/L of lead do not need to be replaced and can be counted toward the number of LSLs required to be replaced.

The State of Wisconsin has primary enforcement responsibility for administering the LCR because it has adopted regulations that are at least as stringent as the federal regulations. *See* Wisconsin Administrative Code [insert code citation]. The State regulation currently applies to all CWSs and NTNCWSs in Wisconsin.

U.S. EPA has the authority to grant a variance from any treatment technique upon a showing by any person that the alternative treatment technique is at least as efficient in lowering the level of that contaminant in drinking water. Section 1415(a)(3) of SDWA, 42 U.S.C. ' 300g-4(a)(3), provides:

> AThe Administrator may grant a variance from any treatment technique requirement of a national primary drinking water regulation upon a showing by any person that an alternative treatment technique not included in such requirement is at least as efficient in lowering the level of the contaminant with respect to which such requirement was prescribed. A variance under this paragraph shall be conditioned on the use of the alternative treatment technique which is the basis for the variance.@

See also 40 C.F.R. ' 142.46.

2. Factual Background

U.S. EPA and the Wisconsin Department of Natural Resources (WDNR) have agreed on the need to better integrate implementation of the statutory and regulatory requirements under the Clean Water Act (CWA)and Safe Drinking Water Act (SDWA) to protect public health and improve our nation's environment. Therefore, the U.S. EPA and WDNR have agreed to establish a more effective approach to reducing the lead levels in drinking water which would also reduce the phosphorus loadings in Wisconsin waters. The U.S. EPA and WDNR have concluded that successful projects demonstrate that in some cases, changes in U.S. EPA regulations, policies, guidance, or interpretations are needed to improve upon the nation's existing public health and environmental protection system. Where such changes can be made under existing law, U.S. EPA agrees to initiate the process for making the changes -- following applicable procedures.

The LCR requires that all systems optimize corrosion control to minimize lead and copper levels at consumers' taps. Many systems currently utilize orthophosphate as the primary lead and copper corrosion control mechanism and the addition of orthophosphate has been effective at reducing lead and copper levels in drinking water under the SDWA. The allowable discharge limits for phosphorus into receiving waters are being lowered under the CWA in Wisconsin such that the amount of orthophosphate being added as part of the Optimal Corrosion Control Treatment (OCCT) for SDWA compliance would require certain entities under the CWA to install treatment to remove the phosphorus prior to being able to

discharge into receiving waters, even where they have added none of the phosphorus themselves (e.g., PWS plant discharges and potable water used in non-contact cooling water applications that is discharged to receiving waters). Almost all lead and copper comes from plumbing materials transporting drinking water to the homes via the distribution system and from plumbing within the homes themselves, therefore there is no possibility to remove these contaminants at the drinking water treatment plant.

A SDWA ban on the use of leaded solder and other leaded materials became effective in 1988 with subsequent additions and modifications to the law since then. It is no longer permissible to install most leaded materials in potable water applications within a public water system or premise plumbing. While the SDWA prohibits the introduction of most leaded materials into the plumbing network, it does not require the removal of existing lead sources. Lead service lines (LSLs), leaded brass and to a more limited extent leaded solder continue to leach lead into the drinking water, with the largest contributor overall being LSLs. The available options for effectively reducing lead and copper levels in PWSs with LSLs without the use of orthophosphate are very limited and could require significant additional water quality and operational changes, including capital improvements.

Many of the same entities regulated under both the CWA and SDWA must comply with lead in drinking water reductions under the SDWA and phosphorus discharge limits under the CWA. To accomplish this, a PWS with LSLs may be required to increase the level of orthophosphate necessary to control lead and copper corrosion at the drinking water plant and to also install treatment to remove the same orthophosphate they have added to the drinking water prior to being able to discharge into receiving waters under the CWA.

The Wisconsin Department of Natural Resources (WDNR) has proposed an alternative treatment technique for compliance with the LCR. WDNR believes that this alternative treatment technique will be more efficient than the LCR treatment technique in lowering lead and copper levels. WDNR proposes that this alternative treatment technique be allowed for certain Public Water Systems (PWSs) in Wisconsin that meet specific criteria. The alternative treatment technique specified in this variance contains a number of provisions, including the permanent removal of all LSLs, including all privately-owned portions of LSLs, within a PWS to lower the levels of lead in the drinking water, along with a corresponding re-evaluation of existing State OCCT designations, with the intent of modifying the State-designated OCCT to eliminate or reduce the level of orthophosphate addition to the water supply once all LSLs,

including all privately-owned portions of LSLs, have been removed from the PWS.

U.S. EPA, Region 5, has reviewed WDNR=s proposal and believes that the proposal has merit and that the alternative treatment technique will be at least as efficient in lowering the level of lead and/or copper in drinking water as the existing treatment technique under the LCR.

U.S. EPA has identified a variance, pursuant to Section 1415(a)(3) of SDWA, 42 U.S.C. ' 300g-4(a)(3), as the appropriate legal mechanism for providing the regulatory flexibility which WDNR has requested. The variance allows certain PWSs to use the alternative treatment technique where specific conditions are met, in lieu of specific LCR requirements. The variance establishes participation criteria that a PWS must meet in order to qualify for the alternative treatment technique. The variance also sets forth the performance criteria that the PWS must meet to continue to be allowed to use this alternative treatment technique. To ensure that the alternative treatment technique is as effective as possible, and provides at least an equivalent level of protection as the existing regulations, U.S. EPA and WDNR have entered into a Memorandum of Understanding (MOU) describing the roles and responsibilities of each agency in implementing the variance. The MOU provides for oversight criteria, which WDNR will follow, to insure the proper implementation of the variance and the use of this alternative treatment technique.

FINDINGS OF FACTS

1. This matter comes before the Regional Administrator of U.S. EPA, Region 5, on request by WDNR, for a State-wide variance pursuant to Section 1415(a)(3) of SDWA, 42 U.S.C. ' 300g-4(a)(3).

2. Pursuant to Section 1401(4)(A) of SDWA, 42 U.S.C. ' 300f(4)(A), a PWS is a system that provides drinking water to the public for human consumption through pipes or other constructed conveyances, and that has at least 15 service connections or regularly serves an average of at least 25 individuals daily at least 60 days out of the year.

3. A CWS is a PWS which serves at least 15 service connections used by year round residents or regularly serves at least 25 year-round residents.

4. An NTNCWS, is a PWS that is not a CWS, and that regularly serves at least 25 of the same persons over 6 months per year.

5.   Pursuant to Section 1401(1)(A) of SDWA, 42 U.S.C.
     ' 300f(1)(A), because CWSs and NTNCWSs are PWSs, certain
     NPDWRs apply to CWSs and NTNCWSs.

6.   The LCR requires all CWSs and NTNCWSs to comply with the
     regulatory requirements specified at 40 C.F.R. ' 141.80
     through ' 141.91.

7.   WDNR requests that a State-wide variance be granted, allowing
     PWSs meeting specific qualifying criteria to use the
     alternative treatment technique outlined in this variance in
     lieu of complying with specific regulatory provisions outlined
     in the LCR.

CONCLUSIONS OF LAW

1.   Section 1415 (a)(3) of SDWA, 42 U.S.C. ' 300g-4(a)(3), and
     40 C.F.R ' 142.46, authorize the Administrator to grant a
     variance from a treatment technique of an NPDWR:

     A...upon a showing by any person that an alternative
     treatment technique not included in such requirement is
     at least as efficient in lowering the level of the
     contaminant with respect to which such requirement was
     prescribed. A variance under this paragraph shall be
     conditioned on the use of the alternative treatment
     technique which is the basis for the variance.@

2.   The authority to issue SDWA variances for treatment technique
     requirements was delegated to the Regional Administrators on
     June 12, 2000. Delegation 9-69, *Issuance of Variances for
     Treatment Technique Requirements*.

3.   PWSs in Wisconsin will be eligible upon application to and
     approval by WDNR, for this variance only if all of the following
     conditions are satisfied:
     a. The PWS has signed a legally-binding agreement with the
        WDNR to remove all LSLs, including all privately-owned
        portions of LSLs, within no more than [15 years] from the
        date of such agreement, unless a lesser amount of time is
        specified by the WDNR.
     b. Any PWS with LSLs that receives water from a PWS which
        has agreed to participate in this variance must agree to
        participate in this variance.
     c. All participating PWSs must demonstrate to the
        satisfaction of U.S. EPA and the WDNR that they have the
        legal authority to require the removal of all LSLs,
        including all privately-owned portions of LSLs.
     d. PWSs must agree to all terms and conditions outlined in
        sections 4 and 5 of this variance, in the agreement
        established under [section xx] of this variance.

4. The PWS must do all of the following in lieu of complying with the requirements specified in 141.80 through 141.82, 141.84, 141.86, 141.87 and 141.88. The requirements specified in this paragraph (4) constitute the alternative treatment technique:

   a. Maintain OCCT that is in place upon the effective date of this variance, as designated by the State until all LSLs are fully removed and the State has provided written approval to modify the PWS' existing OCCT.

   b. Compile an inventory of all LSLs within the PWS' distribution system, including privately-owned portions of LSLs, using the process described in [section xx or paragraph xx of this variance].

   [LISTNUM AutoList36 \l 1] Provide educational material to property owners and residents with LSLs or portions of LSLs regarding the PWS' planned LSL removal program which includes the following:

   1. The benefits of LSL removal;

   2. Health effects information, including the potential for particulate lead release following LSL removal and flushing instructions to minimize the potential for ingestion of released lead particles;

   3. Information regarding the cost, if any, to homeowners for replacing the privately-owned portion of the LSL, including any financing options available to homeowners;

   4. Conduct diagnostic monitoring for lead and copper at the taps on an annual basis during the months of June through September for the duration of the schedule for LSL removal in lieu of compliance monitoring under 40 CFR 141.86.

   d. Conduct diagnostic monitoring for water quality and process control parameters as specified by the WDNR for the duration of the schedule for LSL removal in lieu of compliance monitoring under 40 CFR 141.87.

   [LISTNUM AutoList38 \l 1] Monitoring for lead and copper at the taps shall be used to assess the effectiveness of LSL removal activities and to provide information to the U.S. EPA, WDNR and the public; this data shall not be used in 90th percentile compliance calculations to trigger additional actions by the PWS under the LCR.

   [LISTNUM AutoList38 \l 1] Monitoring for water quality and process control parameters as specified by WDNR shall be used to assess the effectiveness of OCCT as well as the water quality characteristics at the entry points and within the distribution system; this data shall not be used to determine compliance with the LCR TT for OWQCPs under the LCR.

[LISTNUM AutoList38 \l 1]   Comply with all analytical method requirements in 141.89.

[LISTNUM AutoList38 \l 1]   Comply with all applicable LCR requirements (e.g., recordkeeping and reporting) specified in 141.90 and 141.91 and any additional recordkeeping and reporting requirements specified by WDNR in the agreement specified in [paragraph x].

i.   Remove all LSLs within the public water system, including any portions of LSLs not owned by the PWS not later than [15 years] from signing the agreement in [paragraph xx]

Submit a request to WDNR for the elimination or reduction in orthophosphate levels used for OCCT, including any information requested by WDNR and included in the agreement specified in [paragraph xx].

6.   The actions specified in Paragraph 4 above, will be incorporated into individual agreements between WDNR and each participating PWS specified in [subparagraph xx].

7.   The individual agreements will set the time frames for submitting assessments, demonstrations, sample results, designations, and other actions required by this variance, including any additional requirements specified by WDNR.

8.   WDNR will review and act on all submittals in accordance with its existing PWS oversight program.

9.   U.S. EPA and WDNR have entered into an MOU, which will become effective upon the finalization of this variance, and which describes each agency=s responsibilities regarding the variance and the alternative treatment technique.

10.   Approval for the use of the alternative treatment technique will be determined on a case-by-case basis.

ORDER

It is therefore ordered:

That in consultation with WDNR, the Regional Administrator, U.S. EPA, Region 5, finds that WDNR has made a showing for a variance under Section 1415(a)(3) of SDWA. WDNR=s request for a State-wide variance is granted, subject to the following conditions:

1.   All participating PWSs meet the eligibility criteria outlined in [paragraphs xx and xx] of this variance, above.

2.   All participating PWSs meet the participation criteria outlined in [paragraph xx] of this variance, above. Failure to comply with the participation criteria in [paragraph xx]

will automatically terminate the PWS= eligibility for this variance.

3. This variance shall terminate:

    d. Upon termination of the MOU by either WDNR or U.S. EPA; or

    e. Upon a determination by U.S. EPA or WDNR that the alternative treatment technique no longer provides the same level of public health protection as the requirements under the LCR.

5. In the event that the variance terminates, all PWSs subject to this variance shall be required to comply with all requirements under the LCR.

6. The Regional Administrator shall retain jurisdiction and shall annually review the circumstances pertaining to the variance, and may modify or revoke the variance if any provisions or conditions are not met.

7. Nothing in this Order alters or otherwise affects any requirement applicable under the State law.

Dated:_____   _____
                                  Susan Hedman
                                  Regional Administrator

AWWA Lead and Copper Rule Survey

Introduction

Thank you for responding to AWWA's survey on Lead and Copper Rule (LCR) issues. The survey consists of 20 questions and should not take more than 15 minutes to complete. Participant identification is soley for AWWA's use in analyzing the survey results.

Participation before close-of business on July 19, 2013, would be greatly appreciated.

Within several months, USEPA will provide an opportunity for stakeholders to inform its revision of the LCR. AWWA will relay the information collected through this survey to USEPA and other stakeholders through the agency stakeholder process and use the data collected to evaluate proposed rule revisions.

Several central topics will likely be discussed in the upcoming revisions:

1. What requirements will USEPA set to reduce risk from routine activities that disturb
lead service lines (regardless of ownership)?
2. How will USEPA seek to assure corrosion control treatment is in place and adequate?
3. How will USEPA change current lead and copper compliance monitoring requirements?

The survey questions target aspects of each of these three topics.

Developing a clear picture of where lead pipe remains in use as a portion of lead service lines and document typical optimized corrosion control conditions will be an important part of the agency stakeholder process. Your careful consideration of these questions would be very much appreciated.

Completing the Survey

Respondents that manage multiple systems can base their response on the largest system managed or submit multiple survey responses noting different Public Water System Identification (PWSID) numbers.

Questions should be directed to Clay Cope at ccope@awwa.org or (202) 326-6122.

Please see the image below for lead service line replacement terminology
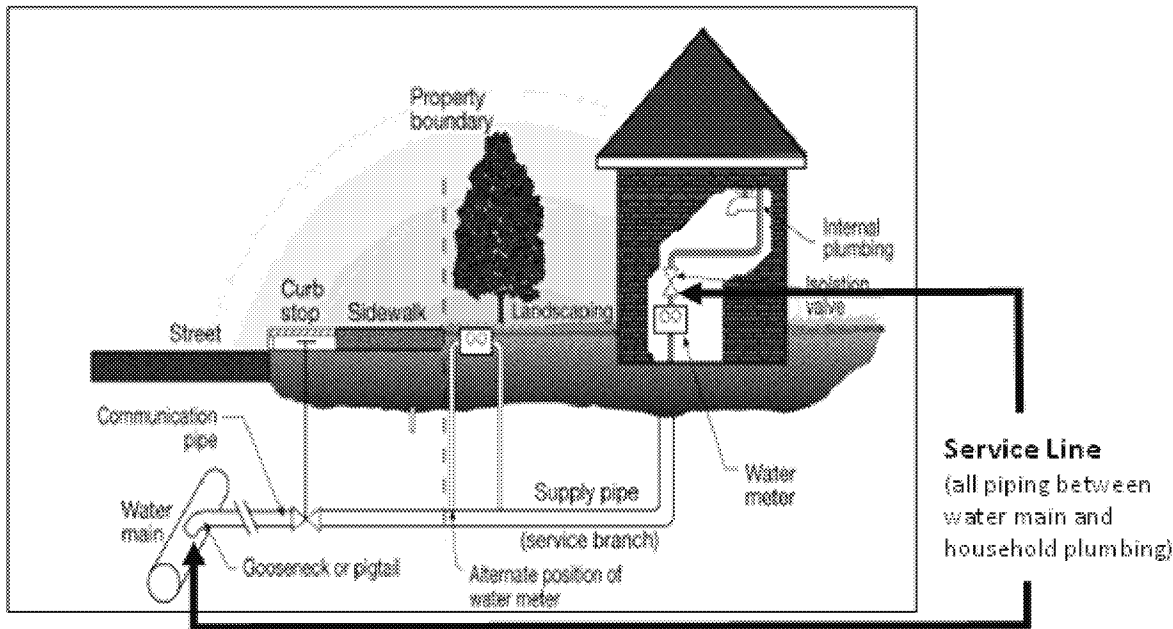
*1. Confirm contact information

Name	
Water System Name	
Phone	
E-mail	
PWSID#	

2. How many service lines in the your water system's service area...?

Contain lead pipe between the water main and curb stop or outside meter (excluding lead goosenecks)

Contain lead pipe between the curb stop or outside meter and house

Contain lead pipe between the water main and meter located inside the house (excluding lead goosenecks)

What is the total number of service lines that have any lead pipe between main and interior plumbing (excluding lead goosenecks)?

What is the total number of service lines that have a lead gooseneck?

3. How many LEAD SERVICE LINES in the the water system's service area, would you estimate, are in each of the following categories?

Owned by customer

Owned by water system

Other

4. Who owns lead service lines located ... ? (check all that apply)

	Owned by water system	Owned by customer	Other
Between the water main and curb stop or outside meter	☐	☐	☐
Between the curb stop or outside meter and house	☐	☐	☐
Between the water main and meter located inside the house	☐	☐	☐

5. What is the basis for the number and location of lead service lines described above? (check all that apply)

☐ Up-to-date, maintained inventory of installations and repairs

☐ Record from initial installations

☐ Historic construction practices

☐ Age of structure served

☐ Dedicated effort to find LSLs

Other (please specify)

6. When undertaking a capital project involving main replacement in several city streets, which of the following does your water system conduct? (check all that apply)

☐ Provide advance notice to the customer

☐ Make an information point of contact available

☐ Provide additional information about possible water quality issues and what to do

☐ Provide additional information about lead and lead service line replacement if encountering lead service lines is likely.

☐ Encourage customers to remove lead service lines they own

☐ Provide water filters to affected customers

Other (please specify)

☐

7. If the water system is replacing water mains in several streets, how does it currently engage and communicate with customers impacted by the distribution system construction activity? (please check all that apply)

☐ Contact with individual customers by phone or in person

☐ Posting information on water system website

☐ Automated telephone contact

☐ E-mail contact

☐ Social media contact

☐ Community meeting near impacted area

☐ Door hangers/postcards attached to door

☐ Direct correspondence

☐ Bill insert with information

☐ Signs posted in area affected

☐ Engage community organizations as partners

Other (please specify)

☐

8. Are there programs available to the system's customers that provide financial assistance that customers could use to undertake full lead service line replacement? (check all that apply):

	This program does not exist in our service area	Water system	Local government agency using community funds	Local government agency using federal program funds	State agency	Local non-profit organization
Cash grant / reimbursal to pay for ENTIRE cost of lead service line replacement (and/or other repairs)	☐	☐	☐	☐	☐	☐
Cash grant / reimbursal to pay for PART of the cost of lead service line replacement (and/or other repairs)	☐	☐	☐	☐	☐	☐
Property tax relief / local tax deduction	☐	☐	☐	☐	☐	☐
Low-interest loan	☐	☐	☐	☐	☐	☐
Extended payment plan incorporated into water bill	☐	☐	☐	☐	☐	☐
Negotiated plumbing service contract with discounted / subsidized rate for service line replacement	☐	☐	☐	☐	☐	☐
Provides complete service line replacement at NO COST	☐	☐	☐	☐	☐	☐
Provides complete service line replacement at REDUCED COST	☐	☐	☐	☐	☐	☐

Other (please specify)

_____

9. Does your water system implement corrosion control to comply with the Lead and Copper Rule?

◯ Yes

◯ No

10. Please describe corrosion control treatment that is currently employed:

What is target pH range for water in the distribution system?

What is target alkalinity range for water in the distribution system (expressed as mg/L as CALCIUM CARBONATE)?

Which secondary disinfectant is used (free chlorine or chloramine)?

What concentration range was reported in last CCR for secondary disinfectant level expressed in mg/L (please note whether value is as FREE CHLORINE or TOTAL CHLORINE)?

If phosphate is applied, what is the target dose range (please specify if PHOSPHORUS or PHOSPHATE)?

11. Which corrosion control inhibitors are currently used by the water system? (check all that apply)

☐ None

☐ Zinc orthophosphate

☐ Phosphoric acid

☐ Polyphosphate

Other (please specify)

12. How is finished water pH adjusted? (select all that apply)

☐ Aeration

☐ Lime

☐ Sodium hydroxide (caustic soda)

☐ Soda ash

Other (please specify)

13. Which of the following is the water system's LCR optimized corrosion control treatment officially based on?

○ Alkalinity and pH adjustment

○ Calcium hardness adjustment

○ Addition of a phosphate inhibitor

○ Addition of a silicate based corrosion inhibitor

○ Other

14. Please indicate any secondary effects that the water system has experienced implementing optimized corrosion control.

	No effect	Positive effect	Negative effect
CT in primary disinfection	○	○	○
Maintaining secondary residual	○	○	○
Managing biofilm growth	○	○	○
Frequency or severity of nitrification	○	○	○
Algae growth in open basins at water treatment plant or in finished water reservoirs	○	○	○
Formation of disinfection by-products (TTHM or HAA5)	○	○	○
Customer complaints	○	○	○
Discolored water	○	○	○
Scale development	○	○	○
Break / leak rate	○	○	○
Loadings at POTW(s) in service area (e.g., nutrients, metals, etc.)	○	○	○

Other (please specify)

[   ]

15. Would your water system be willing to share LCR compliance monitoring data with AWWA? AWWA is collecting LCR compliance monitoring data in order to evaluate potential changes in LCR sampling requirements. If so, please provide name and contact information for the appropriate point-of-contact.

Name	
Phone number	
E-mail address	

16. Who collects your water system's LCR tap samples?

- [ ] Water system customer
- [ ] Dwelling occupant
- [ ] Water system personnel

Other (please specify)

[   ]

17. How many water system staff-hours are involved in collecting a single LCR sample for lead and copper compliance montitoring? (Please include the following in your estimate: recruiting customers to sample, coordinating with state primacy agency, obtaining initial statement of willingness to participate, preparing sample bottles for delivery, delivering sample bottles, recovering sample bottles, documentation and quality control of submitted samples, preparing reports for state primacy agency, and other associated tasks).

[   ]

18. What pipe materials are used in new service line installations and service line repairs in your water system's service area (for service lines 2 inches or smaller in diameter)?

- [ ] Type "K" Copper
- [ ] HDPE
- [ ] PVC SDR 21
- [ ] PVC C 900/905

Other (please specify)

[   ]

19. Does the system observe copper levels above the LCR action level (1.3 mg/L)?

- ( ) Yes
- ( ) No

Evaluation of Gross Alpha and Uranium Measurements for MCL Compliance

Evaluation of Gross Alpha and Uranium Measurements for MCL Compliance

About the Water Research Foundation

The Water Research Foundation (formerly Awwa Research Foundation or AwwaRF) is a member-supported, international, 501(c)3 nonprofit organization that sponsors research to enable water utilities, public health agencies, and other professionals to provide safe and affordable drinking water to consumers.

The Foundation's mission is to advance the science of water to improve the quality of life. To achieve this mission, the Foundation sponsors studies on all aspects of drinking water, including resources, treatment, distribution, and health effects. Funding for research is provided primarily by subscription payments from close to 1,000 water utilities, consulting firms, and manufacturers in North America and abroad. Additional funding comes from collaborative partnerships with other national and international organizations and the U.S. federal government, allowing for resources to be leveraged, expertise to be shared, and broad-based knowledge to be developed and disseminated.

From its headquarters in Denver, Colorado, the Foundation's staff directs and supports the efforts of more than 800 volunteers who serve on the board of trustees and various committees. These volunteers represent many facets of the water industry, and contribute their expertise to select and monitor research studies that benefit the entire drinking water community.

The results of research are disseminated through a number of channels, including reports, the Web site, Webcasts, conferences, and periodicals.

For its subscribers, the Foundation serves as a cooperative program in which water suppliers unite to pool their resources. By applying Foundation research findings, these water suppliers can save substantial costs and stay on the leading edge of drinking water science and technology. Since its inception, the Foundation has supplied the water community with more than $460 million in applied research value.

More information about the Foundation and how to become a subscriber is available on the Web at www.WaterResearchFoundation.org.

Evaluation of Gross Alpha and Uranium Measurements for MCL Compliance

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CONTENTS

TABLES

FIGURES

LIST OF SYMBOLS

Chapter 1

Variable	Definition
$t_{1/2}$	Half-life of a radionuclide
λ	Decay constant of a radionuclide
t	Time
N	Number of radionuclide atoms at time t
N_0	Number of radionuclide atoms at time $t = 0$
A	Activity of a radionuclide at time t at time t
A_0	Activity of a radionuclide at time $t = 0$
A_1	Activity of ^{226}Ra
A_2	Activity of ^{222}Rn
λ_2	Decay constant of ^{222}Rn
A_i	Activity of the ith radionuclide

Chapter 2

Variable	Definition
e	Efficiency of an alpha emitter
C	Detector count rate due to an alpha emitter
A	Activity due to an alpha emitter
e_i	Efficiency of the ith radionuclide
C_T	Detector count rate due to all alpha emitters
C_i	Detector count rate due to the ith alpha emitter
GAA	Gross alpha-particle activity
V	Sample volume
e_S	Average efficiency of the calibration standard
A_i	Activity due to the ith alpha emitter
A_T	Total alpha activity
σ	Measure of radial uniformity of residue
σ_{max}	Maximum value of σ
$\xi(r)$	Radial distribution function of the residue

Chapter 2 (continued)	
Variable	Definition
x	Fraction of the planchet covered by patches
r_1	Radius of the central disc of a nonuniform residue
r_2	Inside radius of the outer ring of a nonuniform residue
r_3	Planchet radius and outside radius of the outer ring of a nonuniform residue
e	Measure of radial nonuniformity of the residue
D	Percent difference between ε_N and the efficiency ε_U
ε_D	Efficiency of the central disc-shaped part of the residue ($r < r_1$)
ε_R	Efficiency of the outer ring-shaped part of the residue ($r_2 < r < r_3$)
ε_N	Efficiency of a nonuniform residue
ε_U	Efficiency of a uniform residue
T_1	Time between sample collection and preparation
T_2	Time between sample preparation and analysis
T_3	Time between sample collection and analysis

Chapters 4 and 5	
Variable	Definition
GAA	Gross alpha-particle activity
A_1	Parent activity of a radionuclide that does not decay appreciably over the sample's lifetime
$A_{1,0}$	Parent activity of a radionuclide that decays appreciably over the sample's lifetime
T_1	Time between sample collection and preparation
T_2	Time between sample preparation and analysis
T_3	Time between sample collection and analysis

Chapter 6	
Variable	Definition
ΔT	Count time interval in minutes
N	Number of counts accumulated in time T
σ_N	Error or standard deviation in N
GAA	Gross alpha-particle activity in pCi/L
σ_{GAA}	Error or standard deviation in GAA in pCi/L

Chapter 6 (continued)	
Variable	Definition
e_S	Average efficiency of the calibration standard
V	Sample volume in liters
C	Conversion factor from uranium activity (pCi/L) to concentration (µg/L)
A_2	^{238}U activity
A_1	^{234}U activity

Chapter 7	
Variable	Definition
t	Time
A_1	^{228}Ra activity at time t
$A_{1,0}$	^{228}Ra activity at time $t = 0$
A_4	^{224}Ra activity at time t
λ_1	Decay constant of ^{228}Ra
λ_3	Decay constant of ^{228Th}
c_{41}, c_{43}	Coefficients in the Bateman equations

Chapter 8	
Variable	Definition
T_1	Time between sample collection and preparation
T_2	Time between sample preparation and analysis
T_3	Time between sample collection and analysis
GAA	Gross alpha-particle activity
e_S	Average efficiency of the calibration standard
V	Volume of the sample in liters
ΔT_S	Sample count interval in minutes
N_S	Number of alpha counts detected in count interval ΔT_S
ΔT_B	Background count interval in minutes
$\overline{C}$	Background count rate in min^{-1}
σ_1	Total error in the GAA
GAA_i	The ith value of the GAA in a triplicate of values
$\langle GAA \rangle$	Average among a triplicate of GAA data all taken at the same time T_2

Chapter 8 (continued)	
Variable	Definition
σ_2	Counting error in the GAA

Chapter 9	
Variable	Definition
GRA	Gross radium activity in pCi/L
V	Volume of the sample in liters
γ	Gravimetric yield of $BaSO_4$
ε_1	Efficiency of ^{226}Ra in $BaSO_4$
λ_2	Decay constant of ^{222}Rn
T_2	Time between sample preparation and analysis
C_T	Number of alpha counts per unit time minus the background count rate

Appendix C	
Variable	Definition
A_i	Activity of ith radionuclide in the sample residue
κ_i	Fraction of ith radionuclide that remains in the sample residue
c_j	jth coefficient in the Bateman equations for the decay of ^{226}Ra
ε_i	Efficiency of ith radionuclide in the sample residue
λ_i	Decay constant of ith radionuclide
t	Time elapsed since the ingrowth of ^{226}Ra progeny began
Δt	An increment in t
ΔN	Number of alpha counts in time interval Δt
ε_{ave}	Weighted average of the efficiencies of the ^{222}Rn, ^{218}Po and ^{214}Po
K	Average among κ_2, κ_3, and κ_6 [$K = (\kappa_2 + \kappa_3 + \kappa_6)/3$]
m	Mass of the sample residue
$f(m)$	Function used to fit to a curve to the efficiency versus residue mass data
a_i, b	For $i = 0$ to 5, adjustable parameters in the function $f(m)$
m_1	Transition mass from "thin" to "thick" residues
D	Fractal dimension of the residue surface ($D = 3 - n$)

	Definition
t	Time elapsed since the ingrowth of ^{224}Ra progeny began
A_i	Activity of ith radionuclide in the sample residue
$A_{1,0}$	^{224}Ra activity in the sample residue at $t = 0$
β_i	Branching ratio at the ith radionuclide
κ_i	Fraction of ith radionuclide that remains in the sample residue
λ_i	Decay constant of ith radionuclide
c_{ij}	ijth coefficient in the Bateman equations for the decay of ^{224}Ra
dN/dt	Instantaneous alpha count rate due to ^{224}Ra and its progeny
ε_i	Efficiency of ith radionuclide in the sample residue
k_1	A constant defined as $k_1 = \varepsilon_1 + \kappa_2\varepsilon_2 + \kappa_3\varepsilon_3 + \beta_5 c_{51}\kappa_5\varepsilon_5 + (1 - \beta_5)c_{51}\kappa_6\varepsilon_6$
k_2	A constant defined as $k_2 = -\beta_5 c_{54}\kappa_5\varepsilon_5 + (1 - \beta_5)c_{54}\kappa_6\varepsilon_6$
Δt	An increment in time t
ΔN	Number of counts in the time period Δt
I_i	Integral of the factor $\exp(-\lambda_i t)$ between $t = t$ and $t = t + \Delta t$
$\varepsilon_{ave,1}$	Weighted average of the efficiencies of ^{224}Ra, ^{220}Rn and ^{216}Po
$\varepsilon_{ave,2}$	Weighted average of the efficiencies of ^{212}Bi and ^{212}Po
K_1	Average among $\kappa_1 (= 1)$ κ_2, and κ_3 $[K_1 = (1 + \kappa_2 + \kappa_3)/3]$
K_2	Average among κ_5 and κ_6 $[K_2 = \beta_5\kappa_5 + (1 - \beta_5)\kappa_6]$
ε	Efficiency of an alpha emitter
E	Alpha particle energy of an alpha emitter
a, b	Fit parameters in the equation $\varepsilon = aE + b$

Appendix D

Variable	Definition
ε	Efficiency of an alpha emitter in a $BaSO_4$ residue
e	Efficiency of an alpha emitter in a sample residue of arbitrary composition
S_P	Planchet area
R_1	Range of an alpha particle in $BaSO_4$ (mg/cm^2)

Appendix D (continued)	
Variable	Definition
R_2	Range of an alpha particle in a sample residue (mg/cm^2)
w_i	Weight fraction of ith element in a residue
r_i	Range of ith element in a residue
A_i	Atomic weight of ith element in a residue

Appendix E	
Variable	Definition
e	Efficiency of an alpha emitter in a sample residue of arbitrary composition
$\langle e \rangle$	Value of e averaged over the residue geometry
S_P	Planchet area
τ	"Thickness" of a sample residue ($\tau = m/S_P$)
τ_0	Average value of τ for a sample residue
$\varphi(\tau)$	$\varphi(\tau)d\tau$ is the probability of a residue having a thickness between τ to $\tau + d\tau$
R_1	Range of an alpha particle in BaSO$_4$ (mg/cm^2)
R_2	Range of an alpha particle in a sample residue (mg/cm^2)
δ	Dirac delta function
$\langle G \rangle$	Value of G averaged over the residue geometry
$\langle \Delta N \rangle$	Value of ΔN averaged over the residue geometry

Appendix F	
Variable	Definition
e_i	Efficiency of ith alpha emitter in a sample residue
N_i	Number of counts due to ith alpha emitter detected in time interval Δt
A_i	Activity of ith alpha emitter in a sample residue
Δt	Sample count interval
A_T	Total activity of all alpha emitters in a sample residue
V	Sample volume
G	Gross alpha-particle activity (GAA) of a sample
ΔN	Total number of alpha counts collected by the detector in time interval Δt
e_S	Average efficiency of the calibration standard
$\langle G \rangle$	Value of G averaged over the residue geometry

Appendix F (continued)

Variable	Definition
$\langle N_i \rangle$	Value of N_i averaged over the residue geometry
$\langle \Delta N \rangle$	Sum of all of the $\langle N_i \rangle$; i.e., $\langle \Delta N \rangle = \langle \Delta N_1 \rangle + \langle \Delta N_2 \rangle + \langle \Delta N_3 \rangle + \dots$
e_S	Average efficiency of the calibration standard
$e_{1,P}$	Efficiency of the calibration standard in a patch residue
$e_{1,S}$	Efficiency of the calibration standard in a smooth residue
$\langle G \rangle$	Value of G due to ^{234}U averaged over the residue geometry
A_1	^{234}U activity
$\langle e \rangle$	Value of the ^{234}U efficiency, e, averaged over the residue geometry
e_S	Average efficiency of the calibration standard
V	Volume of a sample
$e_{1,P}$	Efficiency of the calibration standard in a patch residue
$e_{1,S}$	Efficiency of the calibration standard in a smooth residue
e_1	^{234}U efficiency in a patch residue
e_2	^{234}U efficiency in a smooth residue
$\langle G \rangle$	Value of G due to ^{238}U averaged over the residue geometry
A_1	^{238}U activity
$\langle e \rangle$	Value of the ^{238}U efficiency, e, averaged over the residue geometry
e_S	Average efficiency of the calibration standard
V	Volume of a sample
T_2	Time between sample preparation and analysis
ΔT_2	An increment in T_2
ΔN	Number of alpha counts in time interval ΔT_2
$\langle \Delta N \rangle$	ΔN averaged over the residue geometry
A_1	^{226}Ra activity
λ_2	Decay constant of ^{222}Rn
$\langle e_1 \rangle$	^{226}Ra efficiency, e_1, averaged over the residue geometry
$\langle K_1 e_{ave,1} \rangle$	^{226}Ra progeny efficiency, $K_1 e_{ave,1}$, averaged over the residue geometry
$\langle G \rangle$	G due to ^{226}Ra and progeny averaged over the residue geometry
e_S	Average efficiency of the calibration standard

Appendix F (continued)

V	Sample volume
T_3	Time between sample collection and analysis
A_1	^{210}Po activity at time T_3
$A_{1,0}$	^{210}Po activity at time $T_3 = 0$
λ_1	Decay constant of ^{210}Po
$\langle G \rangle$	Value of G due to ^{210}Po averaged over the residue geometry
$\langle e_1 \rangle$	Value of the ^{210}Po efficiency, e_1, averaged over the residue geometry
e_S	Average efficiency of the calibration standard
V	Sample volume
T_3	Time between sample collection and analysis
A_3	^{210}Po activity
$A_{1,0}$	^{210}Pb activity at $T_3 = 0$
λ_1	Decay constant of ^{210}Pb
λ_2	Decay constant of ^{210}Bi
λ_3	Decay constant of ^{210}Po
c_{ij}	Constants in the Bateman equations for the decay of ^{210}Pb
$\langle G \rangle$	Value of G due to ^{210}Po averaged over the residue geometry
$\langle e_3 \rangle$	Value of the ^{210}Po efficiency, e_3, averaged over the residue geometry
e_S	Average efficiency of the calibration standard
V	Sample volume
T_3	Time between sample collection and analysis
ΔT_3	An increment in T_3
$\langle \Delta N \rangle$	Value of ΔN averaged over the residue geometry
$A_{1,0}$	^{224}Ra activity at $T_3 = 0$
$\langle k_1 \rangle$	Value of k_1 averaged over the residue geometry
$\langle k_2 \rangle$	Value of k_2 averaged over the residue geometry
λ_i	Decay constant of the ith radionuclide
I_i	Integral of the factor $\exp(-\lambda_i t)$ between $t = t$ and $t = t + \Delta t$
c_{ij}	Coefficients of the Bateman equation for the decay of ^{224}Ra

Appendix F (continued)

Variable	Definition
$\langle K_1 e_{\text{ave},1} \rangle$	Value of the $K_1 e_{\text{ave},1}$ averaged over the residue geometry
$\langle K_2 e_{\text{ave},2} \rangle$	Value of the $K_2 e_{\text{ave},2}$ averaged over the residue geometry
$\langle G \rangle$	Value of G due to ^{224}Ra and its progeny averaged over the residue geometry
e_S	Average efficiency of the calibration standard
V	Sample volume
T_3	Time between sample collection and analysis
ΔT_3	An increment in T_3
$\langle G_4 \rangle$	Value of G due to ^{212}Pb progeny averaged over the residue geometry
$A_{4,0}$	^{212}Pb activity at $T_3 = 0$
$\langle K_2 e_{\text{ave},2} \rangle$	Value of the $K_2 e_{\text{ave},2}$ averaged over the residue geometry
q	A constant equal to 1.105
e_S	Average efficiency of the calibration standard
V	Sample volume
T_3	Time between sample collection and analysis
ΔT_3	An increment in T_3
A_i	Activity of ith radionuclide in the sample residue
$A_{1,0}$	^{228}Ra activity at $T_3 = 0$
κ_i	Fraction of ith radionuclide that remains in the sample residue
β_i	Branching ratio of ith radionuclide
λ_i	Decay constant of ith radionuclide
c_{ij}	ijth coefficient in the Bateman equations for the decay of ^{224}Ra
$\langle \Delta N_1 \rangle$	Number of ^{228}Th alpha counts in time interval ΔT_3 averaged over residue geometry
$\langle \Delta N_2 \rangle$	Number of ^{224}Ra, ^{220}Rn, and ^{216}Po alpha counts in the time interval ΔT_3 averaged over residue geometry
$\langle \Delta N_3 \rangle$	Number of ^{212}Bi and ^{212}Po alpha counts in the time interval ΔT_3 averaged over residue geometry
$\langle \Delta N \rangle$	Number of alpha counts due to ^{228}Ra progeny $\langle \Delta N \rangle = \langle \Delta N_1 \rangle + \langle \Delta N_2 \rangle + \langle \Delta N_3 \rangle$
$\langle K_1 e_{\text{ave},1} \rangle$	Value of the $K_1 e_{\text{ave},1}$ averaged over the residue geometry
$\langle K_2 e_{\text{ave},2} \rangle$	Value of the $K_2 e_{\text{ave},2}$ averaged over the residue geometry

Appendix F (continued)	
$\langle G \rangle$	Value of G due to ^{228}Ra progeny averaged over the residue geometry
e_S	Average efficiency of the calibration standard
V	Sample volume

Appendix G	
Variable	**Definition**
ε	Efficiency of the sample residue
l	Thickness of the sample residue
z	The z-coordinate of a point in the sample residue
$\eta(z)$	Function that gives the efficiency of the residue at the plane $z = z$.
m	Mass of the residue

Appendix H	
Variable	**Definition**
N_2^D	Number of ^{220}Rn atoms in the detector region
t	Time elapsed since sample was placed in the instrument
Δt	An increment in t
F	Rate at which ^{220}Rn atoms escape from the BaSO$_4$ residue
$A_{1,0}$	Activity of ^{224}Ra at $t = 0$
e	Proportionality constant between F and the ^{224}Ra activity of the residue
λ_i	Decay constant of ith radionuclide
f	Fraction of the ^{220}Rn atoms that remain in the detector region
ΔN^D	Number of alpha counts detected in time interval Δt
c_{ij}	Coefficients in the Bateman equations for the decay of ^{224}Ra
ε	Efficiency with which the contaminating alpha emitters register detector counts

Appendix I	
Variable	**Definition**
A_T	Total activity of ^{234}U and ^{238}U in a sample
m_T	Total mass of ^{234}U and ^{238}U in a sample
C	Conversion factor between uranium activity (pCi/L) and concentration (μg/L)
λ_1	Decay constant of ^{234}U

λ_2	Decay constant of ^{238}U
N_1	Number of ^{234}U atoms
N_2	Number of ^{238}U atoms
A_1	^{234}U activity
A_2	^{238}U activity

Appendix J

Variable	Definition
G	The gross-alpha-particle activity in pCi/L
ΔT_S	Sample count interval in minutes
ΔT_B	Background count interval in minutes
N_S	Number of alpha counts detected in count interval ΔT_S
$N_{B,i}$	For the ith background count, the number of counts in the count interval ΔT_B
$\overline{C}$	Background count rate in min^{-1}
σ_2	Counting error in the GAA in pCi/L
σ_{N_S}	Standard deviation or error in N_S
$\sigma_{\overline{C}}$	Standard deviation or error in $\overline{C}$ in min^{-1}
V	Volume of the sample in liters
e_S	Efficiency of the calibration standard

FOREWORD

The Water Research Foundation (Foundation) is a nonprofit corporation that is dedicated to the implementation of a research effort to help utilities respond to regulatory requirements and traditional high-priority concerns of the industry. The research agenda is developed through a process of consultation with subscribers and drinking water professionals. Under the umbrella of a Strategic Research Plan, the Research Advisory Council prioritizes the suggested projects based upon current and future needs, applicability, and past work; the recommendations are forwarded to the Board of Trustees for final selection. The Foundation also sponsors research projects through the unsolicited proposal process; the Collaborative Research, Research Applications, and Tailored Collaboration programs; and various joint research efforts with organizations such as the U.S. Environmental Protection Agency, the U.S. Bureau of Reclamation, and the Association of California Water Agencies.

This publication is a result of one of these sponsored studies, and it is hoped that its findings will be applied in communities throughout the world. The following report serves not only as a means of communicating the results of the water industry's centralized research program but also as a tool to enlist the further support of the nonmember utilities and individuals.

Projects are managed closely from their inception to the final report by the Foundation's staff and large cadre of volunteers who willingly contribute their time and expertise. The Foundation serves a planning and management function and awards contracts to other institutions such as water utilities, universities, and engineering firms. The funding for this research effort comes primarily from the Subscription Program, through which water utilities subscribe to the research program and make an annual payment proportionate to the volume of water they deliver and consultants and manufacturers subscribe based on their annual billings. The program offers a cost-effective and fair method for funding research in the public interest.

A broad spectrum of water supply issues is addressed by the Foundation's research agenda: resources, treatment and operations, distribution and storage, water quality and analysis, toxicology, economics, and management. The ultimate purpose of the coordinated effort is to assist water suppliers to provide the highest possible quality of water economically and reliably. The true benefits are realized when the results are implemented at the utility level. The Foundation's trustees are pleased to offer this publication as a contribution toward that end.

David E. Rager   Robert C. Renner, P.E.
Chair, Board of Trustees   Executive Director
Water Research Foundation   Water Research Foundation

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Palm and Pine RV Park
Felda, FL

Oak Park Mobile Home Village
Saint Alva, FL

Petross Water System
Vidalia, GA

North Georgia Water Systems, Inc.
Watkinsville, GA

Fairways Home Owners Association
Caldwell, ID

Joliet Public Works and Utilities Department
Joliet, IL
Romeoville Public Works
Romeoville, IL

Lehigh City Water Plant
Lehigh, IA

Alliance Water Resources
Maquoketa, IA

City of Burdett
Burdett, KS

Stonington Water Company
Stonington, ME

Charles County Department of Utilities
La Plata, MD

Taneytown Utilities Department
Taneytown, MD

Woodstock Job Corps Center
Woodstock, MD

Village of Chatham
Chatham, MI

Mason Department of Public Works
Mason, MI

City of Arlington
Arlington, MN

City of Claremont Water Department
Claremont, MN

New Richland Maintenance Department
New Richland, MN

City of Taconite
Taconite, MN
s
City of Fromberg
Fromberg, MT

Columbia Water and Light Department
Columbia, MO

McCaul, McCaul, and Associates
Potosi, MO

Grand Island Utilities Department
Grand Island, NE

Municipal Light & Water
North Platte, NE

F. X. Lyons, Inc. Water & Pump Service
Intervale, NH

GPM Associates
Cherry Hill, NJ

U. S. Geological Survey
New Jersey Water Science Center
West Trenton, NJ

Sierra Estates GID
Carson City, NV

United States Geological Survey
Nevada Water Science Center
Carson City, NV

South Carolina Department of Health and
Environmental Control
Columbia, SC

South Dakota Department of Environment and
Natural Resources
Pierre, SD

South Dakota Geological Survey
Rapid City, SD

South Dakota Geological Survey
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South West Water Company
Houston, TX

United States Geological Survey
Texas Water Science Center
Shenandoah, TX

Allouez Village Water Department
Village of Allouez, WI

City of Fond du Lac Water Utility
Fond du Lac, WI

Gresham Waterworks
Village of Gresham, WI

City of Thorp Public Works & Utilities
Thorp, WI

Waukesha Water Utility
Waukesha, WI

EXECUTIVE SUMMARY

BACKGROUND

Radioactive minerals in an aquifer are the source of radionuclides (radioactive isotopes) in groundwater. In all aquifers, radionuclides can reside in the water, in the solids, and on the solid surfaces. Some isotopes, like ^{224}Ra, may reside in all three phases; some isotopes, like ^{228}Th, mainly reside in the solids and on the solid surfaces and are virtually absent in the water. The radiological composition of a groundwater sample at the time of collection reflects the composition of the groundwater in the aquifer. Once groundwater is removed from an aquifer and is no longer in contact with the minerals, its radiological composition can change substantially over time. Some radionuclides, like ^{224}Ra, decay away in about three weeks. Some, like ^{228}Th, which were absent in the sample at collection time, are produced by other radionuclides, like ^{228}Ra, and can accumulate to a significant level in the sample. Thus, with the passage of time, a sample's radiological composition can progressively diverge from its composition at collection time.

The gross alpha-particle activity (GAA) of a water sample is intended to approximate the total alpha activity of the sample. However, the GAA is subject to various sources of bias and error (discussed in detail in Chapters 2, 4, 5, 6 and 8) that can cause a sample's GAA to be substantially higher or lower than the sample's actual alpha activity and can cause duplicate measurements to differ significantly from one another. Water samples are commonly analyzed for the alpha emitters ^{226}Ra, ^{234}U, and ^{238}U. Frequently, it is believed that the sum of the ^{226}Ra, ^{234}U, and ^{238}U activities should be equal to the GAA, but there is often a large discrepancy between this sum and the GAA.
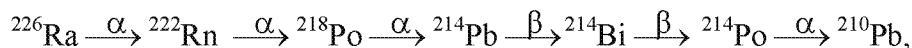
Two types of methods are used to measure a sample's GAA: evaporation methods and coprecipitation methods. In an evaporation method, like EPA Method 900.0 (U.S EPA 1980a), an aliquot of a water sample is evaporated to dryness leaving a relatively thin, solid residue on the bottom of a shallow dish, called a planchet. The residue contains the non-volatile radionuclides of the aliquot. In a coprecipitation method, like Standard Methods 7110C (SM 1998c), the non-volatile radionuclides of an aliquot of water are coprecipitated with a mixture of barium sulfate and ferric hydroxide. The precipitate is collected on a filter, which is placed in a planchet. In either method, the collection of solids in the planchet is called the sample residue. A detector is used to measure the rate at which alpha-particles are emitted from the residue, and the alpha-particle emission rate is used to calculate the GAA.

Two types of samples are used in GAA analyses: grab samples and quarterly composite samples. For a grab sample, the water source is sampled once. For a quarterly composite sample, equal aliquots of water are combined in each of four consecutive three-month periods. The sample holding time for both types of sample is six months. For a quarterly composite sample, the holding time begins when the fourth aliquot is added.

Over the course of the six-month holding time, the radiological composition of a grab sample can vary appreciably and at times bears little resemblance to the original composition. The radiological composition of a quarterly composites sample can vary more than that of a grab sample because the first three aliquots could have been added to the composite sample by as much as nine months prior the fourth aliquot. The graphs of Chapter 4 and the example of Chapter 5 show how the variation of a sample's radiological composition over time affects its GAA,

and show the extent to which the GAA can differ between a grab sample and the corresponding quarterly composite sample.

All of a sample's alpha emitters contribute to its GAA; however, many common alpha emitters are not routinely quantified. Often, a radionuclide, called a parent, produces a series of relatively short-lived radionuclides called its progeny. The parent and progeny constitute a decay chain. The activities of a decay chain's members are not independent but are fixed by the laws of radioactive decay. The following is an example of a decay chain:

$$^{226}\text{Ra} \xrightarrow{\alpha} {}^{222}\text{Rn} \xrightarrow{\alpha} {}^{218}\text{Po} \xrightarrow{\alpha} {}^{214}\text{Pb} \xrightarrow{\beta} {}^{214}\text{Bi} \xrightarrow{\beta} {}^{214}\text{Po} \xrightarrow{\alpha} {}^{210}\text{Pb},$$

where ^{226}Ra is the parent and the subsequent radionuclides are the progeny. Here, "α" denotes an alpha emitter, a radionuclide that emits an alpha particle, and "β" denotes a beta emitter, a radionuclide that emits a beta particle. ^{222}Rn, ^{218}Po, and ^{214}Po are relatively short-lived, alpha-emitting progeny whose contribution to the GAA is frequently ignored; however, whenever the parent, ^{226}Ra, is present, it continually produces progeny, and, in such cases, the short-lived progeny can substantially contribute to the GAA. All of the decay chains of importance in this study are enumerated in Chapter 1.

Any sample that contains a significant ^{228}Ra activity will usually contain a comparable ^{224}Ra activity at the time of collection. ^{224}Ra, a parent, in turn, produces four alpha emitters—^{220}Rn, ^{216}Po, ^{22}Bi, and ^{212}Po—that all contribute to the GAA. The ^{224}Ra activity of a sample is rarely determined and often ignored, but if a sample has a significant ^{228}Ra activity and is analyzed within one week of collection, ^{224}Ra and its progeny often account for most of the sample's GAA.

Even when all of a sample's alpha emitters have been accounted for, the sum of the alpha activities rarely equals the GAA. An alpha emitter's contribution to the GAA is measured by its efficiency, which is the fraction of its alpha particles that reach the detector. The efficiency increases as the alpha-particle energy increases. Many high-energy alpha emitters—like ^{226}Ra progeny, ^{224}Ra and its progeny, and ^{228}Ra progeny—are not routinely quantified but often account for most of the GAA. If a sample contains significant amounts of the high-energy alpha emitters, its GAA often exceeds its alpha activity.

The GAA is equal to the sample residue's alpha-particle count rate divided by the efficiency of a calibration standard:

$$\text{GAA} = \frac{\text{alpha count rate}}{\text{standard efficiency}}.$$

Natural uranium and ^{230}Th are commonly used as calibration standards. In addition, ^{241}Am is used in some coprecipitation methods. The alpha-particle energies, and efficiencies, of the calibration standards are not equal but increase in the order

$$\text{natural uranium} < {}^{230}\text{Th} < {}^{241}\text{Am}.$$

Thus, a sample's GAA will depend on which calibration standard was used, and the GAA will increase in the order

$$^{241}\text{Am} < {}^{230}\text{Th} < \text{natural uranium}.$$

A sample's GAA could be under the maximum contaminant level (MCL) when ^{241}Am is used and over the MCL when ^{230}Th is used.

It is frequently stated that a sample's GAA is the sum of the activities of its alpha emitters excluding uranium and radon. This statement often causes confusion because the way in which radon is "excluded" differs from the way in which uranium is "excluded." Radon is purportedly excluded because it does not precipitate with the sample residue. However, once the residue forms, the radon isotopes ^{220}Rn and ^{222}Rn, produced by ^{224}Ra and ^{226}Ra, are trapped in the residue. The ^{220}Rn activity is maximized within 10 minutes of residue formation, making it impossible to exclude. While the initial ^{222}Rn activity is negligible, once the residue forms, the ^{222}Rn activity steadily increases until it equals the ^{226}Rn activity, which takes about 23 days. Frequently, both ^{220}Rn and ^{222}Rn contribute to the GAA and both produce additional alpha emitters that contribute to the GAA. Thus, in radium-containing samples, radon is never entirely excluded from the GAA.

A sample's uranium activity is "excluded" from its GAA by subtracting its uranium activity concentration (pCi/L) from its GAA to get the adjusted GAA:

$$\text{Adjusted GAA} = \text{GAA} - \text{uranium activity concentration.}$$

The MCL for the adjusted GAA is 15 pCi/L.

Some uranium methods, like fluorometric, laser phosphorimetric, and ICP-MS methods, give an accurate uranium mass concentration but tend to underestimate the uranium activity concentration, which may cause the adjusted GAA to be overestimated and lead to a false-positive GAA violation. A uranium MCL violation occurs when the mass concentration of the uranium isotopes—^{234}U, ^{235}U, and ^{238}U—exceeds 30 µg/L. Some uranium methods, like radiochemical methods, give an accurate uranium activity concentration (pCi/L) but tend to overestimate the uranium mass concentration (µg/L), which may cause a false-positive uranium violation; Some uranium methods, like alpha spectroscopy and some ICP-MS methods, can accurately measure both; however, none of these ICP-MS methods have as yet been approved by the EPA. Problems with uranium methods are discussed in detail in Chapter 6.

The GAA, like any radiological measurement, is subject to counting error. Counting error is inherent in the measurement and can cause the measured value of the GAA to be greater than or less than the true value. If the true value is near the 15 pCi/L MCL, counting error can cause the GAA to exceed or to be less than the MCL. A detailed discussion of the counting error is presented in Chapter 6.

OBJECTIVES

The objectives of this project were to determine and quantify the source of bias and error that affect the gross alpha-particle activity (GAA) and the uranium concentration of drinking water samples, to determine conditions under which the GAA and the uranium concentration can exceed their true values and cause false-positive violations, to provide guidance to water utility, regulatory and laboratory personnel on how to identify the source of a GAA violation and on how to identify and respond to a false-positive violation, and to suggest ways of modifying some of the current radiological methods to improve inter-laboratory consistency. Much experimental and theoretical effort was devoted to the factors affecting the GAA measurement. Less effort was devoted to the factors affecting the uranium measurement, since these are generally well understood.